

US009187544B2

# (12) United States Patent

## Popel et al.

# (10) Patent No.: US 9,187,544 B2

## (45) **Date of Patent:** Nov. 17, 2015

#### (54) PEPTIDE MODULATORS OF ANGIOGENESIS AND USE THEREOF

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(\*) Notice: Subject to any disclaimer, the term of this

patent is extended or adjusted under 35

U.S.C. 154(b) by 0 days.

(21) Appl. No.: 13/962,383

(22) Filed: Aug. 8, 2013

(51) Int CI

#### (65) Prior Publication Data

US 2014/0100164 A1 Apr. 10, 2014

#### Related U.S. Application Data

- (62) Division of application No. 12/522,042, filed as application No. PCT/US2008/000036 on Jan. 3, 2008, now Pat. No. 8,507,434.
- (60) Provisional application No. 60/878,579, filed on Jan. 3, 2007.

(31)	mı. Cı.	
	A61K 38/00	(2006.01)
	A61K 38/10	(2006.01)
	C07K 7/00	(2006.01)
	C07K 7/08	(2006.01)
	C07K 14/575	(2006.01)
	C07K 14/00	(2006.01)
(52)	U.S. Cl.	
. ,	CPC <i>C071</i>	K 14/57518 (2013.01); C071
		(2012.01) 16177.20/00/02

(58) Field of Classification Search

CPC ........ A61K 38/00; A61K 38/10; C07K 7/00; C07K 7/08
USPC ....... 514/13.3, 19.3, 21.5; 530/327
See application file for complete search history.

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#### (57) ABSTRACT

Compositions and methods that are useful for modulating blood vessel formation, as well as methods that provide for the systematic and efficient identification of angiogenesis modulators, are described. As described in more detail below, a systematic computational methodology based on bioinformatics was used to identify novel peptide modulators of angiogenesis that have been characterized in vitro and/or in vivo.

#### 7 Claims, 22 Drawing Sheets

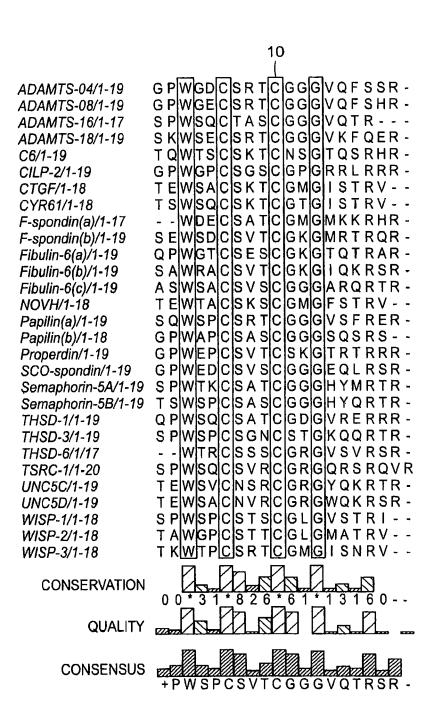
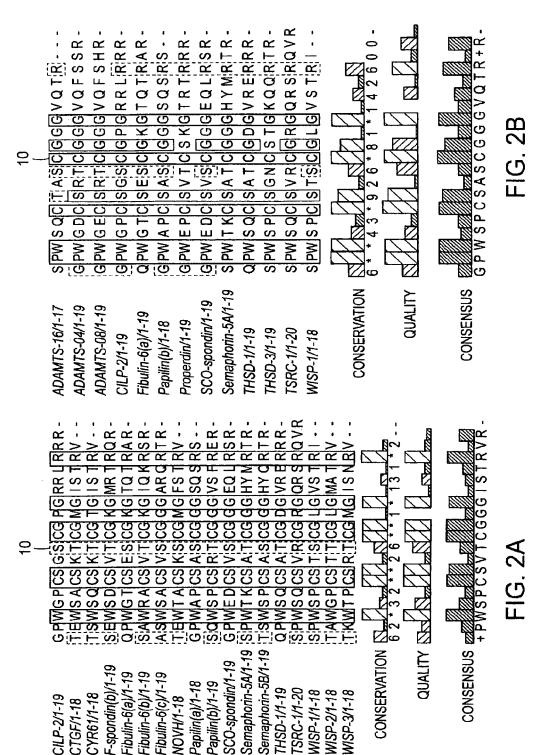
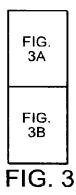


FIG. 1





10ر GPWGDCSRTCGGGVQYTMR-ADAMTS-01/1-20 ADAMTS-02/1-18 GPIWIS QICIS V TICIGNIGITQER - - -GPIWIS EICIS V TICIG EIGIT E V R - - -ADAMTS-03/1-18 GPWGDCSRTCGGGV-----ADAMTS-04(a)/1-15 GPWGDCSRTCGGGVQFSSR-ADAMTS-04(b)/1-20 GPWL ACSRTCDTGWHTR - - -ADAMTS-05/1-18 QPIWIS EICIS ATICIA GIGIV - - - - -ADAMTS-06(a)/1-15 ADAMTS-06(b)/1-18 QPIWIS EICIS ATICIA GIGIVQRQ - - -GPWGQCSGPCGGGVQRR - - -ADAMTS-07(a)/1-18 GPWT KICT VTCGRIGIV - - - - -ADAMTS-07(b)1-15 GPWG ECSRTCGGGGV-----ADAMTS-08(a)1-15 GPWG ECSRTCGGGGVQFSHR-ADAMTS-08(b)1-20 - - WIS SICIS V TICIG QIGIRATR - - -ADAMTS-09(a)1-16 ADAMTS-09(b)1-18 GPIWIG AICIS STICIA GIGISQRR - - -T PIWIG DICISRTICIGGIGIVSSSSR-ADAMTS-10/1-20 - - WD LCSTSCGGGFQKR - - -ADAMTS-12(a)/1-16 SPIWIS HICLSRTICIGALGIV - - - - -ADAMTS-12(b)/1-15 - - WM ECS VSCGDG 1 Q RR - - -ADAMTS-13/1-16 - - WIS QICIS ATICIGEIGIIQQR - - -ADAMTS-14/1-16 SAWS PCSKSCGRGFQRR - - -ADAMTS-15/1-18 ADAMTS-16(a)/1-18 SPIWIS QICITASICIGGIGIVQTR - - -SPIWIS QICITASICIGGIGIVQTRS - -ADAMTS-16(b)/1-19 - PIWIQ QICIT V TICIG GIGI V Q T R - - -ADAMTS-18(a)/1-17 - PWQ QCTVTCGGGGVQTRS - -ADAMTS-18(b)/1-18

FIG. 3A

## FIG. 3B

```
ADAMTS-18(c)/1-18 GPWSQCSKTCGRGVRKR - - -
ADAMTS-18(d)/1-20 SKIWISEICISRTICIGGIGIVKFQER -
                  - - WISKICIS I TICIG KIGIMQSRV - -
ADAMTS-19/1-17
ADAMTS-20(a)/1-18 NS|W|NE|C|S VT|C|G S|G|VQQR - - -
ADAMTS-20(b)/1-19 GPWGQCS SSCSGGLQHRA--
ADAMTS-20(c)/1-16 - - | W | SK | C | S V T | C | G I | G | I M K R - - -
                  SPIWISVICISSTICIGEIGIWQTRTR -
BAI-1/1-20
                  SPIWISVICIS LTICIG QIGILQ VRTR -
BAI-2/1-20
                  SPWSLCSFTCGRGQRTRTR -
BAI-3/1-19
                  TOWITSICIS KTICINSIGITQSRHR -
C6/1-20
CILP-2/1-20
                  GPIWIGPICIS GSICIGPIGIRRLRRR -
                  TEWISAICISKTICIGMIGIISTRV - -
CTGF/1-19
                  TSIWISQICISKTICIGTIGIISTRV - -
CYR61/1-19
                  - - | W | DE | C | SAT | C | GM | G | M K K R H R -
F-spondin(a)/1-18
                  SEWISDICIS VTICIG KIGIMRTRQR -
F-spondin(b)/1-20
                  SAWRACS VTCGKGIQKRSR -
Fibulin-6(a)/1-20
                  QP|W|GT|C|SES|C|GK|G|TQTRAR-
Fibulin-6(b)/1-20
                  A SIWIS AICIS V SICIG GIGIA R Q R T R -
Fibulin-6(c)/1-20
                  TEWITACISKSICIGMIGIFSTRV - -
NOVH/1-19
                  SQWISPICISRTICIGGIGIVSFRER -
Papilin(a)/1-20
                  GP|W|AP|C|SAS|C|GG|G|SQSRS---
Papilin(b)/1-19
                  GPIWIEPICIS VTICISKIGITRTRRR -
Properdin/1-20
                  GPWEDICIS VSICIGGIGEQLRSR -
SCO-spondin/1-20
                  QPWSQCSATCGDGVRERRR -
THSD-1/1-20
                  SPIWISPICIS GNICIS TIGIKQQR TR -
THSD-3/1-20
                  - - WITRICIS SSICIGRIGIVSVRSR -
THSD-6/1-18
                  SPWSSICIS VTICIG VIGIN I TRIR -
TSP-2(a)/1-20
                  SPIWISAICIT VTICIA GIGI I RERTR -
TSP-2(b)/1-20
                  SPWSQCSVRCGRGQRSRQVR
TSRC-1/1-20
                  TELWISVICIN SRICIGRIGIYQKRTR -
UNC5C/1-20
                  TEWSAICIN VRICIGRIGIWQKRSR -
UNC5D/1-20
                  SPIWISPICISTSICIGLIGIVSTR 1 - -
WISP-1/1-19
                  TAWGPCSTTCGLGMATRV--
WISP-2/1-19
                  TKWTPCSRTCGMGISNRV - -
WISP-3/1-19
     CONSERVATION 0 0
       CONSENSUS Z
```

GPWSQCSVTCGGGVQTRSRR

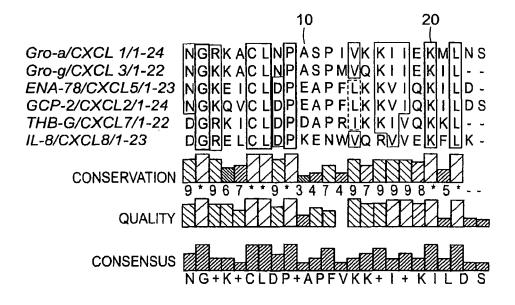
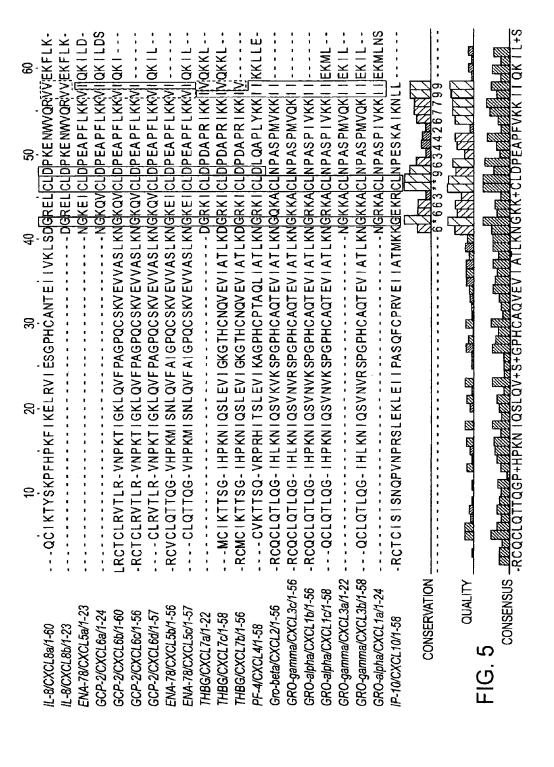


FIG. 4



10 20 30 40 50 60 70	)		QEKAHNQDLGLAGSCLARFST	QEKAHNQDLGLAGSCLPVFSTLP	)	9 ** 6 3 7 * * 5 9 3 1 3 3 4 2		S <u>«ENTON-TOTOR—TOTOR DEPENDENT OF THE FORMANCE OF THE STAP STAP STAP STAP STAP STAP STAP STAP</u>
	alpha1CIVc/1-20 alpha5CIVc/1-20 alpha1CIVa/1-78 alpha1CIVb/1-20	alpha5CIVb/1-20 alpha5CIVa/1-78 alpha6CIVa/1-77	alpha2CIVa/1-77 alpha2CIVa/1-77 alpha2CIVb/1-20	alpha4CIVa/1-77 alpha4CIVb/1-20	alpha4CIVc/1-20	CONSERVATION	QUALITY	CONSENSUS

FIG. 6A

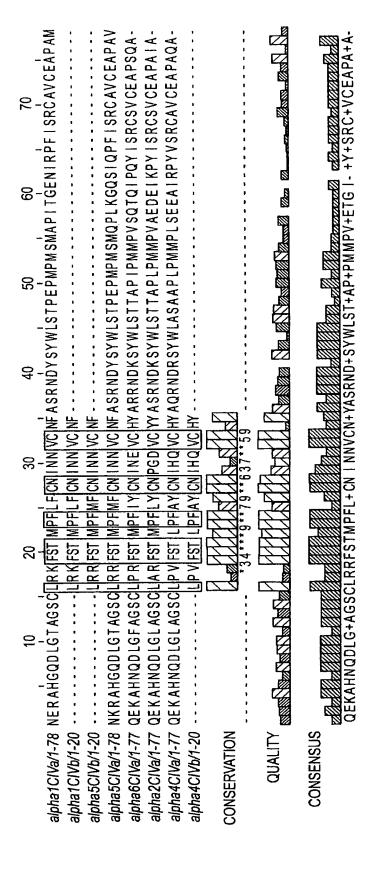


FIG. 68

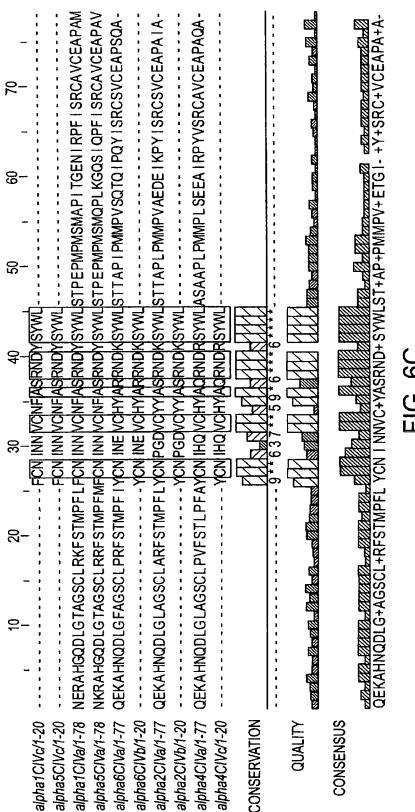
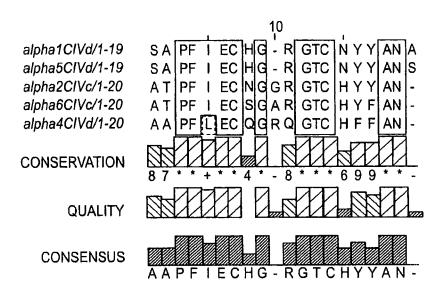


FIG. 6C



A less common motif within the sequences of collagen derived peptide fragments.

FIG. 7

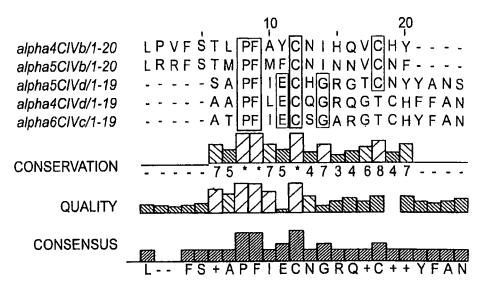
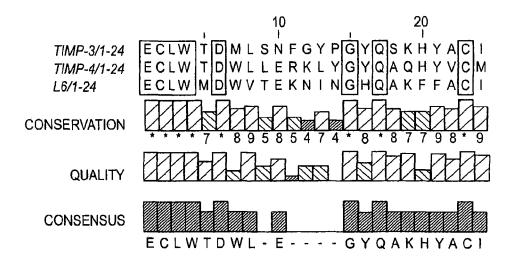


FIG. 8



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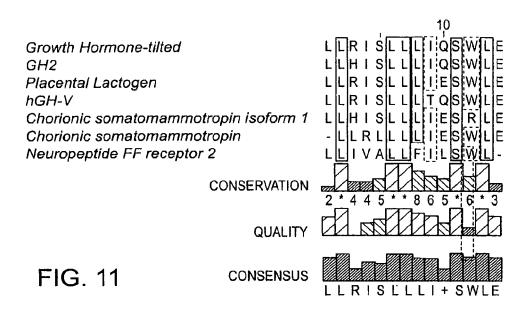
FIG. 9

	Growth Hormone-tilted	LLRIISILL IQ - SWLE
Somatotropins	Placental Lactogen	
	hGH-V	LILIRIISILIL LITQ - SWLE
	Chorionic somatomammotropin-like1	LLHISLL IE - SRLE
	Transmembrane protein 45A	LLRSSLIIILQGSWF-
	Chorionic somatomammotropin	L LRL LL  E- SW L E
	Neuropeptide FF receptor 2	LLIVALLEIL - SWL -
	Brush border myosin-1	LMRKSQILIS-SWF-
	IL-17 receptor C	- R L R - [L][L] T L[Q] - [S W][L] L

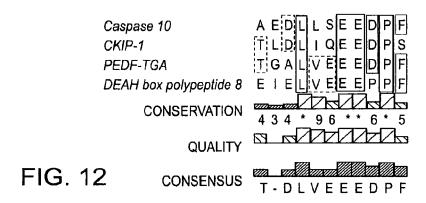
FIG. 10A

Caspase 10 CKIP-1 TGALVEEEDPF EIELVEEEPPF PEDF-TGA DEAH box polypeptide 8

FIG. 10B



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		10
	Collagen IV(a1)	LRKFSTMPFL FCNTNNVCNF
	Collagen IV(a5)	LRRF STMP FM F CN INNV CN F
	Collagen IV(a3)	LQRFIT MPFILF CNVNDVCNF
	Collagen IV(a6)	LPRFSTMPFIY CNINEVCHY
EIO 40	Collagen IV(a2)	LARFSTMPFLYCNPGDVCYY
FIG. 13	Collagen IV(a4)	LPVFSTLPFAYCNIHQVCHY

**CXC Derived Peptide** Human Sequence LRRFSTMPFMFCNINNVCNF GPWEPCSVTCSKGTRTRRR Collagen IV Derived Peptide TSP1 Derived Peptide

NGRKACLNPASPIVKKIIEKMLNS

NGREACLDPEAPLYQKIVQKMLKG LRRFSTMPFMFCNINNVCNF GPWGPCSVTCSKGTQIRQR

Modifications

Mouse Sequence

C substituted by Abu, S, A

Pegylation

Disulfide Bond Formation

M substituted by I K substituted by R

ningetinl fq-itnA Anti-ανβ3 Integrin FIG. 15B FIG. FIG. Concentration Concentration 0 µg/ml Antibody Antibody Collagen Derived Peptides 1001 40, 20. 8 80 40, Ś 80, 60 Peptide Concentration (µg/ml) Peptide Concentration (µg/ml) 100 တ္ထ 99 40 8 Ġ 40 2 vtivitoA ebitqe¶ % § % % 4 √ 9 % Peptide Activity

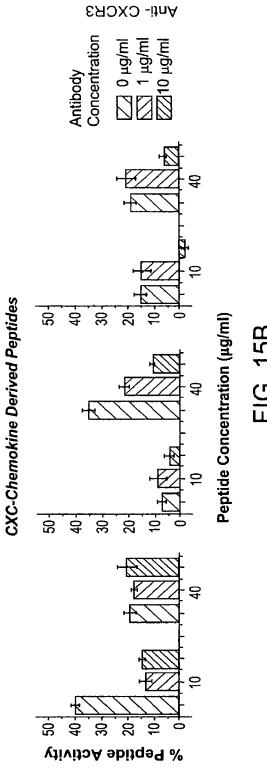
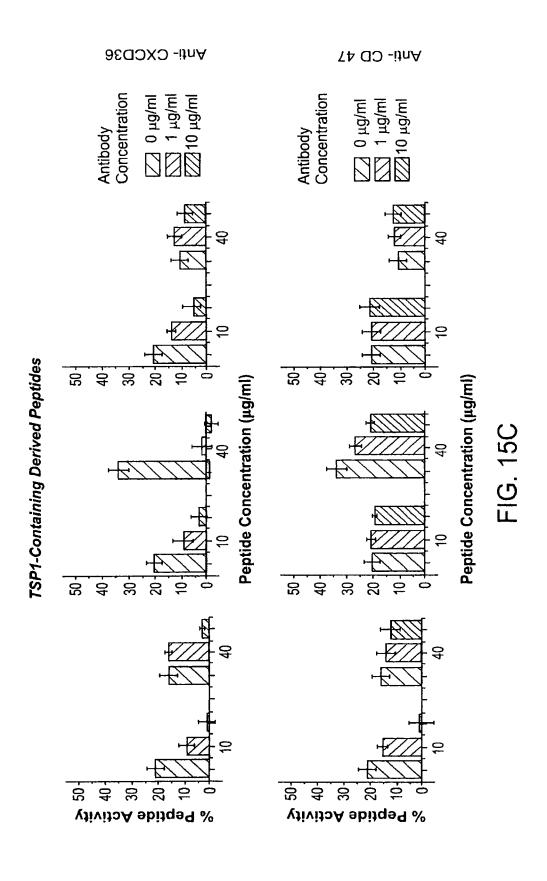
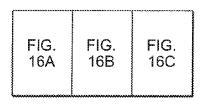


FIG. 15B





TSP1 & Collagen IV

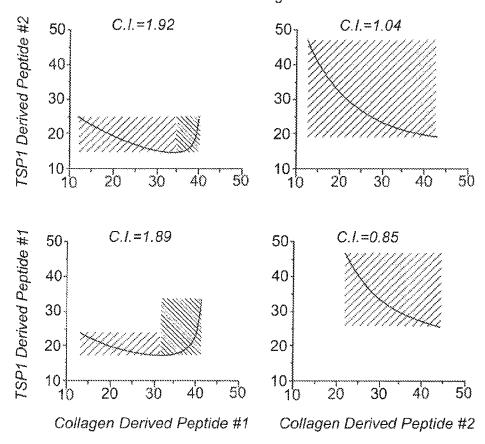


FIG. 16A

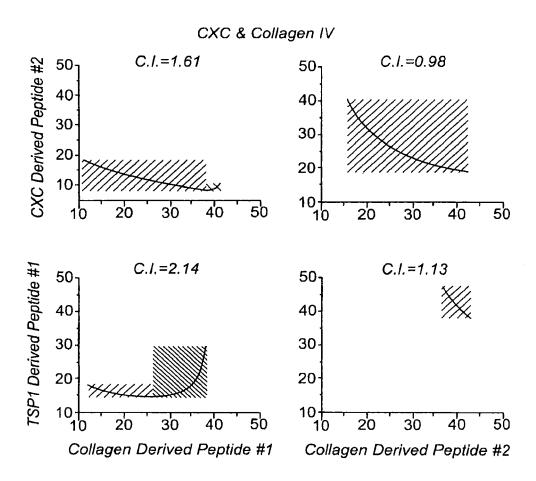


FIG. 16B

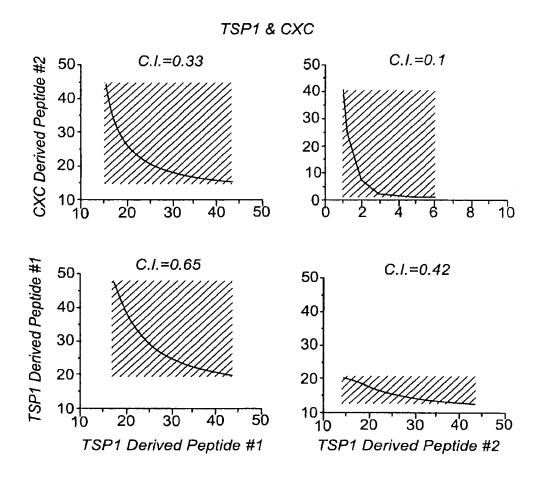
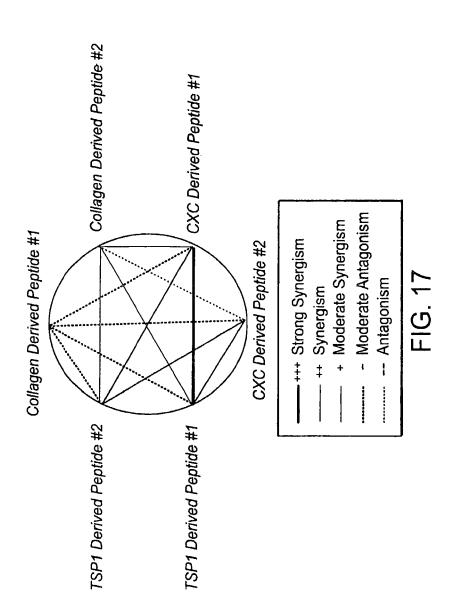
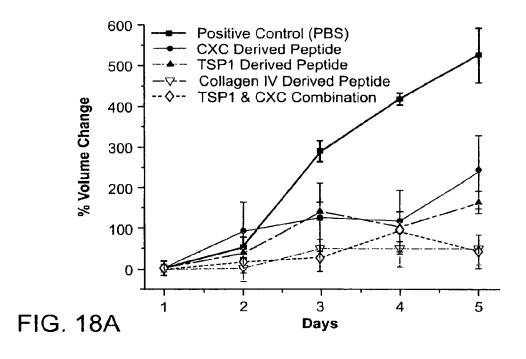


FIG. 16C





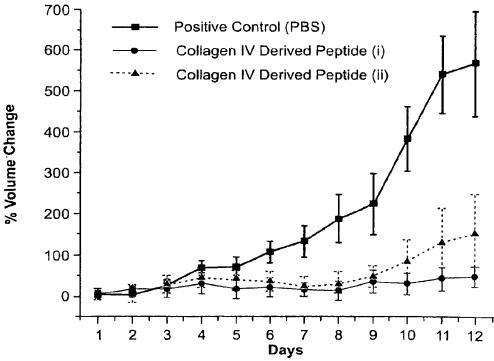


FIG. 18B

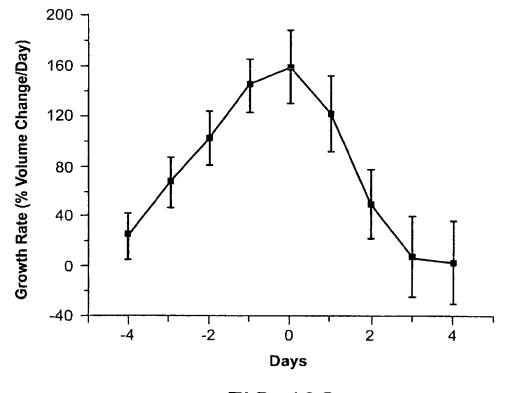


FIG. 18C

#### PEPTIDE MODULATORS OF ANGIOGENESIS AND USE THEREOF

#### CROSS-REFERENCE TO RELATED APPLICATION

This application is a divisional of U.S. application Ser. No. 12/522,042, filed Sep. 22, 2010 which is the U.S. national phase application, pursuant to 35 U.S.C. §371 of PCT International Application Ser. No. PCT/US2008/000036, filed Jan. 3, 2008, designating the United States and published in English, which claims priority to U.S. provisional patent application Ser. No. 60/878,579, filed on Jan. 3, 2007, the entire contents of the aforementioned patent applications are 15 where X denotes a variable amino acid and the number in incorporated herein by this reference.

#### STATEMENT OF RIGHTS TO INVENTIONS MADE UNDER FEDERALLY SPONSORED RESEARCH

This work was supported by the following grant from the National Institutes of Health, Grant No.: HL079653 and CA103175. The government may have certain rights in the invention.

#### SEQUENCE LISTING

The instant application contains a Sequence Listing which has been submitted in ASCII format via EFS-Web and is 30 hereby incorporated by reference in its entirety. Said ASCII copy, created on Aug. 8, 2013, is named 67101DIV\_ 71699 ST25.txt and is 376,832 bytes in size.

#### BACKGROUND OF THE INVENTION

Angiogenesis, the process of developing a novel vascular network from a pre-existing one, is tightly controlled by various endogenous regulators. These regulatory elements include both pro- and anti-angiogenic proteins that finely 4 modulate the neovascular morphological and functional characteristics. Where the regulation of such processes is disrupted a variety of pathological conditions can ensue, including neoplasia, hematologic malignancies, rheumatoid arthritis, diabetic retinopathy, age-related macular degenera- 4 tion, atherosclerosis, endometriosis, pathologic obesity, and ischemic heart and limb disease. An urgent need exists for angiogenesis modulators that can be used as therapeutics for these and other numerous angiogenesis related diseases and conditions. While some promising angiogenesis modulators 5 have been identified, to date, the quest for the experimental identification of such agents has been an empirical timeconsuming process. Improved angiogenesis modulators and methods for systematically identifying and assessing the biological activity of such agents are urgently required.

#### SUMMARY OF THE INVENTION

As described below, the present invention generally features angiogenesis modulators, related prophylactic and 6 therapeutic methods, as well as screening methods for the identification of such agents.

The invention generally provides peptides that reduce blood vessel formation in a cell, tissue, or organ. Accordingly, in one aspect the invention features an isolated peptide or 65 analog thereof containing one of the following amino acid sequences:

2

		(CEO ID NO	0007
	TSP Motif:	(SEQ ID NO: W-X(2)-C-X(3)-C-X(2)-G	2287)
5	CXC Motif:	G-X(3)-C-L	
		(SEQ ID NO:	2288)
	Collagen Motif:	C-N-X(3)-V-C	
	Collagen Motif:	P-F-X(2)-C	
10	Somatotropin Motif:	$( \mbox{SEQ ID NO:} \\ \mbox{L-X(3)-L-L-X(3)-S-X-L}$	2289)
	Serpin Motif:	$\label{eq:continuous} \text{(SEQ ID NO: } \\ \text{L-X(2)-E-E-X-P;}$	2290)

parentheses denotes the number of variable amino acids; W denotes tryptophan; C denotes cysteine, G denotes glycine, V denotes valine; L denotes leucine, P is proline, and where the peptide reduces blood vessel formation in a cell, tissue or 20 organ. In one embodiment, the peptide contains an amino acid sequence shown in Table 1-6, 8 and 9. In yet another embodiment, the peptide further contains at least 5, 10, 15, or 20 amino acids flanking the naturally occurring sequence.

In another aspect, the invention features an isolated peptide  $^{25}\,$  or analog thereof having at least 85%, 90%, 95%, or 100% identity to an amino acid sequence shown in Table 1-10 or otherwise disclosed herein. In one embodiment, the peptide contains an amino acid sequence shown in Table 1-10. In another embodiment, the peptide consists essentially of an amino acid sequence shown in Table 1-10. In yet another embodiment, the peptide further contains at least 5, 10, 15, or 20 amino acids flanking the naturally occurring sequence.

In yet another aspect, the invention features an isolated peptide or analog thereof containing or consisting essentially of a sequence having at least 85% 90%, 95%, or 100% amino acid sequence identity to an amino acid sequence selected from the group consisting of:

40	Placental Lactogen	(SEQ ID NO: 2291) LLRISLLLIESWLE
	hGH-V	(SEQ ID NO: 2292) LLRISLLLTQSWLE
45	GH2	(SEQ ID NO: 2293) LLHISLLLIQSWLE
	Chorionic somatomammotropin	(SEQ ID NO: 2294) LLRLLLLIESWLE
50	Chorionic somatomammotro- pin hormone-like 1	(SEQ ID NO: 2295) LLHISLLLIESRLE
55	Transmembrane protein 45A	(SEQ ID NO: 2296) LLRSSLILLQGSWF
	IL-17 receptor C	(SEQ ID NO: 2297) RLRLLTLQSWLL
60	Neuropeptide FF receptor 2	(SEQ ID NO: 2298) LLIVALLFILSWL
	Brush border myosin-I	(SEQ ID NO: 2299) LMRKSQILISSWF

where the peptide reduces blood vessel formation in a cell, tissue or organ.

In yet another aspect, the invention features an isolated peptide or analog thereof containing or consisting essentially

of a sequence having at least 85%, 90%, 95%, or 100% amino acid sequence identity to an amino acid sequence selected from the group consisting of:

DEAH		(SEQ	ID	NO:	2300)
box polypep- tide 8	EIELVEEEPPF	(SEQ	ID	NO:	2301)
Caspase 10	AEDLLSEEDPF	(SEQ	ID	NO:	2302)
CKIP-1	TLDLIQEEDPS	(SEQ	ID	NO:	2303)

where the peptide reduces blood vessel formation in a cell, tissue or organ.

In yet another aspect, the invention features an isolated peptide or analog thereof containing or consisting essentially of a sequence having at least 85% amino acid sequence identity to an amino acid sequence selected from the group consisting of:

where the peptide reduces blood vessel formation in a cell, tissue or organ.

In another aspect, the invention features a pharmaceutical composition containing an effective amount of an isolated peptide containing an amino acid sequence shown in Table 1-10 or a peptide analog thereof in a pharmacologically acceptable excipient. In one embodiment, the composition 35 contains at least one peptide that is a TSP, CXC, Collagen IV, Somatotropin, or Serpin derived peptide. In another embodiment, the composition contains at least two, three, four, or five peptides selected from the group consisting of TSP, CXC, Collagen IV, Somatotropin, and Serpin derived peptides. In 40 one embodiment, the composition contains at least a CXC derived peptide and a TSP1 derived peptide. In another embodiment, the CXC derived peptide contains the amino acid sequence NGRKACLNPASPIVKKIIEKMLNS (SEQ ID NO: 2305). In yet another embodiment, the TSP1 repeat- 45 containing protein contains the amino acid sequence GPWEPCSVTCSKGTRTRRR (SEQ ID NO: 2306).

In a related aspect, the invention features an isolated nucleic acid molecule encoding the peptide of any previous aspect.

In another related aspect, the invention features an expression vector containing the nucleic acid molecule of the previous aspect, where the nucleic acid molecule is positioned for expression. In one embodiment, the vector includes a promoter suitable for expressing the nucleic acid molecule in 55 a mammalian cell.

In yet another related aspect, the invention features a host cell containing the peptide of any previous aspect or a nucleic acid molecule encoding the peptide. In one embodiment, the cell is a prokaryotic or eukaryotic cell (e.g., mammalian, 60 human). In another embodiment, the cell is in vitro or in vivo.

In another aspect, the invention features a method of reducing blood vessel formation in a tissue or organ, the method involving contacting an endothelial cell, or a tissue or organ containing an endothelial cell with an effective amount of a 65 peptide of any previous aspect, thereby reducing blood vessel formation in the tissue or organ.

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In yet another aspect, the invention features a method of reducing endothelial cell proliferation, migration, survival, or stability in a tissue or organ, the method involving contacting tissue or organ containing an endothelial cell with an effective amount of a peptide of any previous aspect.

In still another aspect, the invention features a method of increasing endothelial cell death in a tissue or organ, the method involving contacting a tissue or organ containing an endothelial cell with an effective amount of a peptide of any o previous aspect, thereby increasing endothelial cell death in the tissue or organ.

In another aspect, the invention features a method of reducing blood vessel formation in a tissue or organ the method involving contacting the tissue, or organ with a vector encoding a peptide of any previous aspect; and expressing the peptide in a cell of the tissue or organ, thereby reducing blood vessel formation in the tissue or organ.

In another aspect, the invention features a method of modulating angiogenesis in a cell, tissue, or organ, the method involving contacting the cell, tissue, or organ with an effective amount of an agent that binds CD36, CD47 or CXCR3.

In another aspect, the invention features a method for treating a neoplasia in a subject in need thereof, the method involving administering an effective amount of a peptide of any previous aspect. In one embodiment, at least one peptide binds CD36, CD47 or CXCR3. In another embodiment, the method involves administering two peptides, one that binds CD36 or CD47 and one that binds CXCR3. In yet another embodiment, the method reduces angiogenesis in a neoplastic tissue. In yet another embodiment, the neoplasia is lung carcinoma.

In another aspect, the invention features a kit containing an effective amount of a peptide of any previous aspect, and directions for using the peptide to treat a disease characterized by undesirable or excess angiogenesis.

In various embodiments of any of the above aspects, the peptide contains a motif delineated herein or an amino acid sequence delineated herein. In various embodiments of the above aspects, the peptide contains an alteration in one amino acid relative to a reference sequence shown in Tables 1-10. In various embodiments of the above aspects, the peptide contains at least one modification (e.g., a sequence alteration or post-translational modification that increases protease resistance, biodistribution, or therapeutic efficacy). In various embodiments of the above aspects, the peptide is cyclized or pegylated. In other embodiments delineated herein, the sequence alteration replaces a cysteine with aminobutyric acid (Abu), serine or alanine, replaces methionine with isoleucine, or replaces lysine with arginine. In various embodiments of the above aspects, the peptide contains at least 10, 20, 30, 40, or 50 amino acids of a naturally occurring amino acid sequence described by an NCBI reference number listed in Table 1-10. In various embodiments of the above aspects, the tissue or organ is in vitro or in vivo. In other embodiments, the cell is a human cell, tissue, or organ. In yet other embodiments, the cell is a neoplastic cell (lung carcinoma cell). In another embodiment, the method treats a neoplasia (e.g., lung carcinoma). In another embodiment, the method treats corneal or choroidal neovascularization. In another embodiment, the number or volume of blood vessels in the tissue or organ (e.g., mammalian tissue or organ) are reduced by at least 10%, 25%, 30%, 50%, 75% or more relative to a control condition. In another embodiment, the peptide acts on an endothelial cell. In various embodiments of the above aspects, the method involves contacting the cell tissue or organ with two agents, one that binds CD36 or CD47 and one that binds CXCR3 (e.g., a CXC derived peptide or a TSP1 derived peptide). In

another embodiment, the method involves administering at least two peptides, such as a CXC derived peptide and a TSP1 derived peptide; a peptide that binds CD36 or CD47 and a peptide that binds BetaI or BetaIII integrin; a TSP derived peptide and a collagen IV derived peptide. In various embodiments of the above aspects, the method involves administering a combination of two, three, four, or more peptides shown in Table 1-10.

#### **Definitions**

By "analog" is meant a chemical compounds having a structure that is different from the general structure of a reference agent, but that functions in a manner similar to the reference agent. For example, a peptide analog having a variation in sequence or having a modified amino acid.

By "thrombospondin (TSP) derived peptide" is meant a peptide comprising a TSP motif: W-X(2)-C-X(3)-C-X(2)-G (SEQ ID NO: 2287). Exemplary TSP derived peptides are shown in Tables 1 and 2. If desired, the peptide includes at the carboxy or amino terminus of the motif in the naturally occurring amino acid sequence of the peptide. TSP1 derived peptides include, for example, those derived from proteins WISP-1 (SPWSPCSTSCGLGVSTRI (SEQ ID NO: 2307)), NOVH (TEWTACSKSCGMGFSTRV (SEQ ID NO: 2308)) 25 and UNC5C (TEWSVCNSRCGRGYQKRTR (SEQ ID NO: 2309)).

By "CXC derived peptide" is meant a peptide comprising a CXC Motif: G-X(3)-C-L. Exemplary CXC derived peptides are shown in Table 3. If desired, the peptide includes at least 30 about 5, 10, 20, 30, 40, 50 or more amino acids that flank the carboxy or amino terminus of the motif in the naturally occurring amino acid sequence. CXC derived peptides include, for example, those derived from proteins GRO-a/CXCL1 (NGRKACLNPASPIVKKIIEKMLNS (SEQ ID NO: 2305)), 35 GRO-γ/MIP-2β/CXCL3 (NGKKACLNPASPMVQKI-IEKIL (SEQ ID NO: 2310)), and ENA-78/CXCL5 (NGKE-ICLDPEAPFLKKVIQKILD (SEQ ID NO: 2311)).

By "Collagen IV derived peptide" is meant a peptide comprising a C-N-X(3)-V-C (SEQ ID NO: 2288) or P-F-X(2)-C 40 collagen motif. Exemplary collagen IV derived peptides are shown in Table 5. If desired, the peptide includes at least about 5, 10, 20, 30, 40, 50 or more amino acids that flank the carboxy or amino terminus of the motif in the naturally occurring amino acid sequence. Type IV collagen derived peptides 45 include, for example, LRRFSTMPFMFCNINNVCNF (SEQ ID NO: 2312) and FCNINNVCNFASRNDYSYWL (SEO ID NO: 2313), and LPRFSTMPFIYCNINEVCHY (SEQ ID NO: 2304).

comprising a Somatotropin Motif: L-X(3)-L-L-X(3)-S-X-L (SEQ ID NO: 2289). Exemplary somatotropin derived peptides are shown in Table 8. If desired, the peptide includes at least about 5, 10, 20, 30, 40, 50 or more amino acids that flank the carboxy or amino terminus of the motif in the naturally 55 occurring amino acid sequence. Somatotropin derived peptides include, for example, those shown in FIG. 10A.

By "Serpin derived peptide" is meant a peptide comprising a Serpin Motif: L-X(2)-E-E-X-P. Exemplary serpin derived peptides are shown in Table 9. If desired, the peptide includes 60 at least about 5, 10, 20, 30, 40, 50 or more amino acids that flank the carboxy or amino terminus of the motif in the naturally occurring amino acid sequence. Serpin derived peptides include, for example, those shown in FIG. 10B.

By "Beta 1 integrin" is meant a polypeptide that binds a 65 collagen IV derived peptide or that has at least about 85% identity to NP\_596867 or a fragment thereof.

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By "Beta 3 integrin" is meant a polypeptide that binds a collagen IV derived peptide or that has at least about 85% identity to P05106 or a fragment thereof.

By "CD36" is meant a CD36 glycoprotein that binds to a thrombospondin-derived peptide or that has at least about 85% identity to NP\_001001548 or a fragment thereof. CD36 is described, for example, by Oquendo et al., "CD36 directly mediates cytoadherence of Plasmodium falciparum parasitized erythrocytes," Cell 58: 95-101, 1989.

By "CD47" is meant a CD47 glycoprotein that binds to a thrombospondin-derived peptides or that has at least about 85% identity to NP\_000315 or a fragment thereof. CD47 is described, for example, by Han et al., "CD47, a ligand for the macrophage fusion receptor, participates in macrophage multinucleation." J. Biol. Chem. 275: 37984-37992, 2000.

By "CXCR3" is meant a G protein coupled receptor or fragment thereof having at least about 85% identity to NP\_001495. CXCR3 is described, for example, by Trentin et least about 5, 10, 20, 30, 40, 50 or more amino acids that flank 20 al., "The chemokine receptor CXCR3 is expressed on malignant B cells and mediates chemotaxis." J. Clin. Invest. 104: 115-121, 1999.

> By "blood vessel formation" is meant the dynamic process that includes one or more steps of blood vessel development and/or maturation, such as angiogenesis, vasculogenesis, formation of an immature blood vessel network, blood vessel remodeling, blood vessel stabilization, blood vessel maturation, blood vessel differentiation, or establishment of a functional blood vessel network.

> By "angiogenesis" is meant the growth of new blood vessels originating from existing blood vessels. Angiogenesis can be assayed by measuring the total length of blood vessel segments per unit area, the functional vascular density (total length of perfused blood vessel per unit area), or the vessel volume density (total of blood vessel volume per unit volume

> By "vasculogenesis" is meant the development of new blood vessels originating from stem cells, angioblasts, or other precursor cells.

> By "blood vessel stability" is meant the maintenance of a blood vessel network.

> By "alteration" is meant a change in the sequence or in a modification (e.g., a post-translational modification) of a gene or polypeptide relative to an endogeneous wild-type reference sequence.

> By "ameliorate" is meant decrease, suppress, attenuate, diminish, arrest, or stabilize the development or progression

By "antibody" is meant any immunoglobulin polypeptide, By "Somatotropin derived peptide" is meant a peptide 50 or fragment thereof, having immunogen binding ability.

In this disclosure, "comprises," "comprising," "containing" and "having" and the like can have the meaning ascribed to them in U.S. Patent law and can mean "includes," "including," and the like; "consisting essentially of" or "consists essentially" likewise has the meaning ascribed in U.S. Patent law and the term is open-ended, allowing for the presence of more than that which is recited so long as basic or novel characteristics of that which is recited is not changed by the presence of more than that which is recited, but excludes prior art embodiments.

A "cancer" in an animal refers to the presence of cells possessing characteristics typical of cancer-causing cells, for example, uncontrolled proliferation, loss of specialized functions, immortality, significant metastatic potential, significant increase in anti-apoptotic activity, rapid growth and proliferation rate, and certain characteristic morphology and cellular markers. In some circumstances, cancer cells will be in the

form of a tumor; such cells may exist locally within an animal, or circulate in the blood stream as independent cells, for example, leukemic cells.

By "disease" is meant any condition or disorder that damages or interferes with the normal function of a cell, tissue, or organ.

By "an effective amount" is meant the amount required to ameliorate the symptoms of a disease relative to an untreated patient. The effective amount of active compound(s) used to practice the present invention for therapeutic treatment of an angiogenesis-associated disease varies depending upon the manner of administration, the age, body weight, and general health of the subject. Ultimately, the attending physician or veterinarian will decide the appropriate amount and dosage regimen. Such amount is referred to as an "effective" amount.

By "fragment" is meant a portion of a polypeptide or nucleic acid molecule. This portion contains, preferably, at least 10%, 20%, 30%, 40%, 50%, 60%, 70%, 80%, or 90% of the entire length of the reference nucleic acid molecule or 20 polypeptide. A fragment may contain 10, 20, 30, 40, 50, 60, 70, 80, 90, or 100, 200, 300, 400, 500, 600, 700, 800, 900, or 1000 nucleotides or amino acids.

By "isolated nucleic acid molecule" is meant a nucleic acid (e.g., a DNA) that is free of the genes, which, in the naturally 25 occurring genome of the organism from which the nucleic acid molecule of the invention is derived, flank the gene. The term therefore includes, for example, a recombinant DNA that is incorporated into a vector; into an autonomously replicating plasmid or virus; or into the genomic DNA of a 30 prokaryote or eukaryote; or that exists as a separate molecule (for example, a cDNA or a genomic or cDNA fragment produced by PCR or restriction endonuclease digestion) independent of other sequences. In addition, the term includes an RNA molecule which is transcribed from a DNA molecule, as 35 well as a recombinant DNA which is part of a hybrid gene encoding additional polypeptide sequence.

By an "isolated polypeptide" is meant a polypeptide of the invention that has been separated from components that naturally accompany it. Typically, the polypeptide is isolated 40 when it is at least 60%, by weight, free from the proteins and naturally-occurring organic molecules with which it is naturally associated. Preferably, the preparation is at least 75%, more preferably at least 90%, and most preferably at least 99%, by weight, a polypeptide of the invention. An isolated 45 polypeptide of the invention may be obtained, for example, by extraction from a natural source, by expression of a recombinant nucleic acid encoding such a polypeptide; or by chemically synthesizing the protein. Purity can be measured by any appropriate method, for example, column chromatography, 50 polyacrylamide gel electrophoresis, or by HPLC analysis.

By "marker" is meant any protein or polynucleotide having an alteration in expression level or activity that is associated with a disease or disorder.

"By "neoplasia" is meant a disease that is caused by or 55 results in inappropriately high levels of cell division, inappropriately low levels of apoptosis, or both. Solid tumors, hematological disorders, and cancers are examples of neoplasias.

By "operably linked" is meant that a first polynucleotide is 60 positioned adjacent to a second polynucleotide that directs transcription of the first polynucleotide when appropriate molecules (e.g., transcriptional activator proteins) are bound to the second polynucleotide.

By "peptide" is meant any fragment of a polypeptide. Typically peptide lengths vary between 5 and 1000 amino acids (e.g., 5, 10, 15, 20, 25, 50, 100, 200, 250, 500, 750, and 1000).

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By "polypeptide" is meant any chain of amino acids, regardless of length or post-translational modification.

By "promoter" is meant a polynucleotide sufficient to direct transcription.

By "reduce" is meant a decrease in a parameter (e.g., blood vessel formation) as detected by standard art known methods, such as those described herein. As used herein, reduce includes a 10% change, preferably a 25% change, more preferably a 40% change, and even more preferably a 50% or greater change.

By "reference" is meant a standard or control condition.

By "subject" is meant a mammal, including, but not limited to, a human or non-human mammal, such as a bovine, equine, canine, ovine, or feline.

By "substantially identical" is meant a polypeptide or nucleic acid molecule exhibiting at least 50% identity to a reference amino acid sequence (for example, any one of the amino acid sequences described herein) or nucleic acid sequence (for example, any one of the nucleic acid sequences described herein). Preferably, such a sequence is at least 60%, more preferably 80% or 85%, and even more preferably 90%, 95% or even 99% identical at the amino acid level or nucleic acid to the sequence used for comparison.

Sequence identity is typically measured using sequence analysis software (for example, Sequence Analysis Software Package of the Genetics Computer Group, University of Wisconsin Biotechnology Center, 1710 University Avenue, Madison, Wis. 53705, BLAST, BESTFIT, GAP, or PILEUP/ PRETTYBOX programs). Such software matches identical or similar sequences by assigning degrees of homology to various substitutions, deletions, and/or other modifications. Conservative substitutions typically include substitutions within the following groups: glycine, alanine; valine, isoleucine, leucine; aspartic acid, glutamic acid, asparagine, glutamine; serine, threonine; lysine, arginine; and phenylalanine, tyrosine. In an exemplary approach to determining the degree of identity, a BLAST program may be used, with a probability score between  $e^{-3}$  and  $e^{-100}$  indicating a closely related sequence.

"Sequence identity" or "identity" in the context of two nucleic acid or polypeptide sequences includes reference to the residues in the two sequences which are the same when aligned for maximum correspondence over a specified comparison window, and can take into consideration additions, deletions and substitutions. When percentage of sequence identity is used in reference to proteins it is recognized that residue positions which are not identical often differ by conservative amino acid substitutions, where amino acid residues are substituted for other amino acid residues with similar chemical properties (for example, charge or hydrophobicity) and therefore do not deleteriously change the functional properties of the molecule. Where sequences differ in conservative substitutions, the percent sequence identity may be adjusted upwards to correct for the conservative nature of the substitution. Sequences which differ by such conservative substitutions are said to have sequence similarity. Approaches for making this adjustment are well-known to those of skill in the art. Typically this involves scoring a conservative substitution as a partial rather than a full mismatch, thereby increasing the percentage sequence identity. Thus, for example, where an identical amino acid is given a score of 1 and a non-conservative substitution is given a score of zero, a conservative substitution is given a score between zero and 1. The scoring of conservative substitutions is calculated, for example, according to the algorithm of Meyers and Miller, Computer

Applic. Biol. Sci., 4: 11-17, 1988, for example, as implemented in the program PC/GENE (Intelligenetics, Mountain View, Calif., USA).

"Percentage of sequence identity" means the value determined by comparing two optimally aligned sequences over a comparison window, wherein the portion of the polynucleotide sequence in the comparison window may comprise additions, substitutions, or deletions (i.e., gaps) as compared to the reference sequence (which does not comprise additions, substitutions, or deletions) for optimal alignment of the two sequences. The percentage is calculated by determining the number of positions at which the identical nucleic acid base or amino acid residue occurs in both sequences to yield the number of matched positions, dividing the number of matched positions by the total number of positions in the window of comparison and multiplying the result by 100 to yield the percentage of sequence identity.

The term "substantial identity" or "homologous" in their various grammatical forms in the context of polynucleotides 20 means that a polynucleotide comprises a sequence that has a desired identity, for example, at least 60% identity, preferably at least 70% sequence identity, more preferably at least 80%, still more preferably at least 90% and even more preferably at least 95%, compared to a reference sequence using one of the 25 alignment programs described using standard parameters. One of skill will recognize that these values can be appropriately adjusted to determine corresponding identity of proteins encoded by two nucleotide sequences by taking into account codon degeneracy, amino acid similarity, reading frame posi- 30 tioning and the like. Substantial identity of amino acid sequences for these purposes normally means sequence identity of at least 60%, more preferably at least 70%, 80%, 85%, 90%, and even more preferably at least 95%.

Another indication that nucleotide sequences are substan- 35 tially identical is if two molecules hybridize to each other under stringent conditions. However, nucleic acids which do not hybridize to each other under stringent conditions are still substantially identical if the polypeptides which they encode are substantially identical. This may occur, for example, 40 when a copy of a nucleic acid is created using the maximum codon degeneracy permitted by the genetic code. One indication that two nucleic acid sequences are substantially identical is that the polypeptide which the first nucleic acid encodes is immunologically cross reactive with the polypep- 45 tide encoded by the second nucleic acid, although such crossreactivity is not required for two polypeptides to be deemed substantially identical.

An "expression vector" is a nucleic acid construct, generated recombinantly or synthetically, bearing a series of speci- 50 fied nucleic acid elements that enable transcription of a particular gene in a host cell. Typically, gene expression is placed under the control of certain regulatory elements, including constitutive or inducible promoters, tissue-preferred regulatory elements, and enhancers.

A "recombinant host" may be any prokaryotic or eukaryotic cell that contains either a cloning vector or expression vector. This term also includes those prokaryotic or eukaryotic cells that have been genetically engineered to contain the

The term "operably linked" is used to describe the connection between regulatory elements and a gene or its coding region. That is, gene expression is typically placed under the control of certain regulatory elements, including constitutive or inducible promoters, tissue-specific regulatory elements, 65 and enhancers. Such a gene or coding region is said to be "operably linked to" or "operatively linked to" or "operably

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associated with" the regulatory elements, meaning that the gene or coding region is controlled or influenced by the regu-

A "reference sequence" is a defined sequence used as a basis for sequence comparison. A reference sequence may be a subset of or the entirety of a specified sequence; for example, a segment of a full-length cDNA or gene sequence, or the complete cDNA or gene sequence. For polypeptides, the length of the reference polypeptide sequence will generally be at least about 5, 10, or 15 amino acids, preferably at least about 20 amino acids, more preferably at least about 25 amino acids, and even more preferably about 35 amino acids, about 50 amino acids, about 100 amino acids, or about 150 amino acids. For nucleic acids, the length of the reference nucleic acid sequence will generally be at least about 50 nucleotides, preferably at least about 60 nucleotides, more preferably at least about 75 nucleotides, and even more preferably about 100 nucleotides about 300 nucleotides or about 450 nucleotides or any integer thereabout or therebetween.

Methods of alignment of sequences for comparison are well-known in the art. Optimal alignment of sequences for comparison may be conducted by the local homology algorithm of Smith and Waterman, Adv. Appl. Math., 2: 482, 1981; by the homology alignment algorithm of Needleman and Wunsch, J. Mol. Biol., 48: 443, 1970; by the search for similarity method of Pearson and Lipman, Proc. Natl. Acad. Sci. USA, 8: 2444, 1988; by computerized implementations of these algorithms, including, but not limited to: CLUSTAL in the PC/Gene program by Intelligenetics, Mountain View, Calif., GAP, BESTFIT, BLAST, FASTA, and TFASTA in the Wisconsin Genetics Software Package, Genetics Computer Group (GCG), 7 Science Dr., Madison, Wis., USA; the CLUSTAL program is well described by Higgins and Sharp, Gene, 73: 237-244, 1988; Corpet, et al., Nucleic Acids Research, 16:881-90, 1988; Huang, et al., Computer Applications in the Biosciences, 8:1-6, 1992; and Pearson, et al., Methods in Molecular Biology, 24:7-331, 1994. The BLAST family of programs which can be used for database similarity searches includes: BLASTN for nucleotide query sequences against nucleotide database sequences; BLASTX for nucleotide query sequences against protein database sequences; BLASTP for protein query sequences against protein database sequences; TBLASTN for protein query sequences against nucleotide database sequences; and TBLASTX for nucleotide query sequences against nucleotide database sequences. See, Current Protocols in Molecular Biology, Chapter 19, Ausubel, et al., Eds., Greene Publishing and Wiley-Interscience, New York, 1995. New versions of the above programs or new programs altogether will undoubtedly become available in the future, and can be used with the present invention.

Unless otherwise stated, sequence identity/similarity values provided herein refer to the value obtained using the BLAST 2.0 suite of programs, or their successors, using 55 default parameters (Altschul et al., Nucleic Acids Res, 2:3389-3402, 1997). It is to be understood that default settings of these parameters can be readily changed as needed in the future.

As those ordinary skilled in the art will understand, cloned gene(s) in the chromosome or genome of the host cell. 60 BLAST searches assume that proteins can be modeled as random sequences. However, many real proteins comprise regions of nonrandom sequences which may be homopolymeric tracts, short-period repeats, or regions enriched in one or more amino acids. Such low-complexity regions may be aligned between unrelated proteins even though other regions of the protein are entirely dissimilar. A number of low-complexity filter programs can be employed to reduce such low-

complexity alignments. For example, the SEG (Wooten and Federhen, *Comput. Chem.*, 17:149-163, 1993) and XNU (Clayerie and States, *Comput. Chem.*, 17:191-1, 1993) low-complexity filters can be employed alone or in combination.

As used herein, the terms "treat," "treating," "treatment," and the like refer to reducing or ameliorating a disorder and/or symptoms associated therewith. It will be appreciated that, although not precluded, treating a disorder or condition does not require that the disorder, condition or symptoms associated therewith be completely eliminated.

A "tumor," as used herein, refers to all neoplastic cell growth and proliferation, whether malignant or benign, and all precancerous and cancerous cells and tissues.

As used herein, the terms "prevent," "preventing," "prevention," "prophylactic treatment" and the like refer to reducing the probability of developing a disorder or condition in a subject, who does not have, but is at risk of or susceptible to developing a disorder or condition.

#### BRIEF DESCRIPTION OF THE DRAWINGS

FIG. 1 shows a set of amino acid sequences that include a shaded 4-letter motif common in all the experimentally tested TSP-1 containing proteins (SEQ ID NOS 2324-2352, respectively, in order of appearance). At the bottom of the figure is 25 the consensus sequence.

FIGS. **2**A and **2**B show a set of amino acid sequences that included shaded common motifs of the TSP-1 containing peptides using a threshold of 60% (FIG. **2**A, SEQ ID NOS 2329-2331, 2333-2337, 2339, 2338, 2341-2344, 2347 and 30 2350-2352, respectively, in order of appearance) and 45% (FIG. **2**B, SEQ ID NOS 2326, 2324-2325, 2329, 2334, 2339-2342, 2344-2345, 2347 and 2350, respectively, in order of appearance).

FIG. 3 shows a set of amino acid sequences that include a shaded 4-letter motif common in all the theoretically predicted TSP-1 containing proteins (FIG. 3A discloses SEQ ID NOS 2356-2359, 2324, 2360-2365, 2325, 2366-2373, 2326 and 2374-2376, respectively, in order of appearance & FIG. 3B discloses SEQ ID NOS 2377, 2327, 2378-2384, 2328-40 2333, 2335, 2334, 2336-2341, 2344-2346, 2385-2386 and 2347-2352, respectively, in order of appearance). In the red insert the predicted motif is identified within TSP-2 domains as well.

FIG. 4 shows a set of amino acid sequences that include a 45 shaded 6-letter motif common in all the experimentally tested C-X-C containing proteins (SEQ ID NOS 2305, 2310-2311 and 2388-2390, respectively, in order of appearance).

FIG. 5 shows a set of amino acid sequences that include a shaded common motif in all the theoretically predicted antiangiogenic C-X-C containing proteins (SEQ ID NOS 2392, 2390, 2311, 2388, 2393-2397, 2389, 2398-2404, 2310, 2405, 2305 and 2406, respectively, in order of appearance).

FIGS. **6**A-**6**C show a set of amino acid sequences that include in shading the most abundant motif in the theoretically predicted anti-angiogenic type IV collagen derived peptide fragments (FIG. **6**A discloses SEQ ID NOS 2313, 2313, 2408-2409, 2312, 2410-2416 and 2320, respectively, in order of appearance; FIG. **6**B discloses SEQ ID NOS 2408-2409, 2312, 2410, 2418-2419 and 2415-2416, respectively, in order of appearance; & FIG. **6**C discloses SEQ ID NOS 2313, 2313, 2408, 2410, 2418, 2412, 2419, 2414-2415 and 2320, respectively, in order of appearance). Novel motifs occur when the abundant 7-mer is shifted downstream (FIG. **6**B) or upstream (FIG. **6**C).

FIG. 7 shows a set of amino acid sequences that include in shading a less common motif within the sequences of type IV

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collagen derived peptide fragments (SEQ ID NOS 2422-2426, respectively, in order of appearance).

FIG. **8** shows a set of amino acid sequences that include in shading a motif identified within the subset of the of type IV collagen derived short anti-angiogenic peptides (SEQ ID NOS 2416, 2312, 2423, 2426 and 2425, respectively, in order of appearance).

FIG. 9 shows a set of amino acid sequences that include in shading a common motif that occurs in all the predicted anti-angiogenic fragments derived from TIMPs (SEQ ID NOS 2429-2431, respectively, in order of appearance).

FIGS. **10**A and **10**B show the amino acid sequences of eleven novel anti-angiogenic peptides. Sequences in FIG. **10**A are from the somatotropin family (SEQ ID NOS 2433, 2291-2292, 2295, 2434, 2294, 2298-2299 and 2297, respectively, in order of appearance) and those in FIG. **10**B are from the serpin family (SEQ ID NOS 2302-2303, 2435 and 2301, respectively, in order of appearance & 'DEAH' disclosed as SEQ ID NO: 2300).

FIG. 11 shows a set of amino acid sequences that include in shading a motif identified within the similarity hits of the Growth Hormone derived anti-angiogenic peptide (SEQ ID NOS 2433, 2293, 2291-2292, 2295, 2294 and 2298, respectively, in order of appearance).

FIG. 12 shows a set of amino acid sequences that include in shading a motif identified within the similarity hits of the PEDF derived short anti-angiogenic peptide (SEQ ID NOS 2302-2303, 2435 and 2301, respectively, in order of appearance & 'DEAH' disclosed as SEQ ID NO: 2300).

FIG. 13 shows the amino acid sequence of a novel peptide derived from the alpha4 fibril of type IV collagen and its similarities with known peptides (SEQ ID NOS 2409, 2312, 2438, 2304, 2428 and 2416, respectively, in order of appearance). Common amino acids are shaded.

FIG. **14** shows exemplary amino acid sequence modifications (SEQ ID NOS 2442, 2444-2445 and 2439-2441, respectively, in order of appearance).

FIGS. 15A-15C includes a set of graphs showing that likely receptors for peptides identified herein were identified in the HUVEC proliferation assay after neutralization of various receptors associated with anti-angiogenic activity. FIG. 15A shows the effect of  $\beta$ - and  $\alpha v \beta 3$  integrin-neutralizing antibodies on the activity of three collagen IV-derived peptides (red). The collagen derived peptides used in the experiment are derived from the alpha5 fibrils of type IV collagen (LRRFSTMPFMFCNINNVCNF (SEQ ID NO: 2312) and FCNINNVCNFASRNDYSYWL (SEO ID NO: 2313)), and from alpha6 fibrils of type IV collagen (LPRFSTMPFIYC-NINEVCHY (SEQ ID NO: 2304)). FIG. 15B shows the effect of two different concentrations of the CXCR3 receptor-neutralizing antibody on the activity of three CXC chemokinederived peptides (green). The CXC derived peptides used in this experiment are derived from proteins GRO-α/CXCL1 (NGRKACLNPASPIVKKIIEKMLNS (SEQ ID NO: 2305)), (NGKKACLNPASPMVQKI-GRO-γ/MIP-2β/CXCL3 IEKIL (SEQ ID NO: 2310)), and ENA-78/CXCL5 (NGKE-ICLDPEAPFLKKVIQKILD (SEQ ID NO: 2311)).

FIG. 15C shows the effect of CD36 and CD47 receptorneutralizing antibodies on the activity of three thrombospondin-derived peptides (blue). The TSP1 repeat-containing protein derived peptides used in the experiment are derived from proteins WISP-1 (SPWSPCSTSCGLGVSTRI (SEQ ID NO: 2307)), NOVH (TEWTACSKSCGMGFSTRV (SEQ ID NO: 2308)) and UNC5C (TEWSVCNSRCGRGYQKRTR (SEQ ID NO: 2309)).

FIGS. 16A-C depict graphs showing the evaluation of peptide combinations from different protein families. Two pep-

tides from each of three different protein families were combined serially in the proliferation assay, and the efficiency of the peptide combinations was evaluated after calculating the isobolograms and Combination Index for each of the combinations. FIG. **16**A depicts data for TSP1 and Collagen IV. 5 FIG. **16**B depicts data for CXC and Collagen IV. FIG. **16**C depicts data for TSP1 and CXC.

FIG. 17 shows a quantitative description of the peptide combinations. The combinations that induce strong synergism are marked with thicker red lines whereas the combinations that induce antagonism are shown with dotted blue lines.

FIGS. 18A-18C are graphs. FIG. 18A shows the results of the administration of collagen IV, TSP1 and CXC derived peptides, as well as the combination of the TSP1 derived 15 peptide and the CXC derived peptide. Each of the peptides was administered at 20 mg/kg/day i.p. (n=3 per condition). For the combination, the peptides were administered alternately every other day. PBS was administered as a positive control. FIG. 18B shows the effect of the administration of the 20 collagen IV derived peptide on tumor volume. The peptide was administered in an i.p. injection at 10 mg/kg/day for 12 days. Control (n=6); peptide application (green, n=6; red, n=5). These results for n=5 do not include one animal in which the tumor started growing after day 9. FIG. 18C shows 25 the tumor growth rate (% volume change per day) on day 14 after inoculation (day 0 at panel C). Once the tumors reached a volume of approximately 800 mm<sup>3</sup> treatment with a TSP1 derived and CXC derived peptides was started. The peptides were administered alternately every other day at a dose of 10 30 mg/kg. The tumor growth rate dropped to zero after 3 injections.

### DETAILED DESCRIPTION OF THE INVENTION

The invention features compositions and methods that are useful for modulating angiogenesis. The invention is based, at least in part, on the discovery of general peptide motifs that are associated with anti-angiogenic properties of peptides. Angiogenesis

Angiogenesis, which involves the growth or sprouting of new microvessels from pre-existing vasculature, and vasculogenesis, which involves de novo vascular growth, is essential to many physiological and pathological conditions, including embryogenesis, cancer, rheumatoid arthritis, dia- 45 betic retinopathy, obesity, atherosclerosis, ischemic heart and limb disease, and wound healing. Over 70 diseases have been identified as angiogenesis dependent (Carmeliet, Nature, 438:932-6, 2005). Under physiological conditions, the growth of new microvessels is tightly regulated and orches- 50 trated by maintaining a balance between endogenous pro- and anti-angiogenic factors. Tipping the balance of this regulation may lead to either excessive neovascularization, as in cancer, age-related macular degeneration, and rheumatoid arthritis, or insufficient vascularization, as in ischemic heart and limb 55 disease, ischemic brain, and neural degeneration.

Angiogenesis is a complex multistep process that involves interactions between endothelial cells (EC), pericytes, vascular smooth muscle cells, and stromal cells (e.g., stem cells and parenchymal cells). These interactions occur through 60 secreted factors, such as vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), basic fibroblast growth factor (bFGF or FGF-2) and angiopoietins, as well as through cell-cell and cell-extracellular matrix (ECM) interactions. Endothelial cell-ECM interactions regulate 65 numerous processes that are critical for angiogenesis, including endothelial cell migration, proliferation, differentiation

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and apoptosis. Angiogenic processes include network stabilization and remodeling that may involve the recruitment of stromal cells, as well as the pruning of some vessels. In many cases, angiogenesis occurs as a response to hypoxia. A transcription factor called hypoxia-inducible factor, HIF1α, has been demonstrated to act as an oxygen sensor whose activity leads to upregulation of VEGF in parenchymal and stromal cells (Semenza, Physiology (Bethesda), 19:176-82, 2004). VEGF is secreted as a homodimer in the form of several heparin-binding and non-heparin-binding splice-variant isoforms; it diffuses through the interstitial space and can bind to the endothelial cell receptors VEGFR1 and VEGFR2, as well as co-receptors such as Neuropilin-1, thus initiating a signal transduction cascade that leads to endothelial cell proliferation and migration. The production of endothelial cell matrix metalloproteinases, MMPs, increases as a result of endothelial cell activation; MMPs are necessary for selectively clipping the capillary basement membrane and the ECM, which constitute physical barriers to endothelial cell migration and capillary sprouting. MMPs and their associated molecules also play a crucial role in uncovering cryptic sites of the ECM proteins, a number of which have been identified as antiangiogenic (Davis et al., Anat Rec, 268:252-75, 2002; Folkman, Annu Rev Med, 57:1-18, 2006; Rundhaug, J Cell Mol Med, 9:267-85, 2005; Schenk and Quaranta, Trends Cell Biol, 13:366-75, 2003), and in processing cell-surface receptors (Mott and Werb, Curr Opin Cell Biol, 16:558-64, 2004). Diseases Associated with Undesirable Angiogenesis

Where the processes regulating angiogenesis are disrupted, pathology may result. Such pathology affects a wide variety of tissues and organ systems. Diseases characterized by excess or undesirable angiogenesis are susceptible to treatment with therapeutic agents described herein.

Excess angiogenesis in numerous organs is associated with cancer and metastasis, including neoplasia and hematologic malignancies.

Angiogenesis-related diseases and disorders are commonly observed in the eye where they may result in blindness. Such disease include, but are not limited to, age-related macular degeneration, choroidal neovascularization, persistent hyperplastic vitreous syndrome, diabetic retinopathy, and retinopathy of prematurity (ROP).

A number of angiogenesis-related diseases are associated with the blood and lymph vessels including transplant arteriopathy and atherosclerosis, where plaques containing blood and lymph vessels form, vascular malformations, DiGeorge syndrome, hereditary hemorrhagic telangiectasia, cavernous hemangioma, cutaneous hemangioma, and lymphatic malformations.

Other angiogenesis diseases and disorders affect the bones, joints, and/or cartilage include, but are not limited to, arthritis, synovitis, osteomyelitis, osteophyte formation, and HIV-induced bone marrow angiogenesis.

The gastro-intestinal tract is also susceptible to angiogenesis diseases and disorders. These include, but are not limited to, inflammatory bowel disease, ascites, peritoneal adhesions, and liver cirrhosis.

Angiogenesis diseases and disorders affecting the kidney include, but are not limited to, diabetic nephropathy (early stage: enlarged glomerular vascular tufts).

Excess angiogenesis in the reproductive system is associated with endometriosis, uterine bleeding, ovarian cysts, ovarian hyperstimulation.

In the lung, excess angiogenesis is associated with primary pulmonary hypertension, asthma, nasal polyps, rhinitis, chronic airway inflammation, cystic fibrosis.

Diseases and disorders characterized by excessive or undesirable angiogenesis in the skin include psoriasis, warts, allergic dermatitis, scar keloids, pyogenic granulomas, blistering disease, Kaposi's sarcoma in AIDS patients, systemic sclerosis

Obesity is also associated with excess angiogenesis (e.g., angiogenesis induced by fatty diet). Adipose tissue may be reduced by the administration of angiogenesis inhibitors.

Excess angiogenesis is associated with a variety of auto-immune disorders, such as systemic sclerosis, multiple sclerosis, Sjögren's disease (in part by activation of mast cells and leukocytes). Undesirable angiogenesis is also associated with a number of infectious diseases, including those associated with pathogens that express (lymph)-angiogenic genes, that induce a (lymph)-angiogenic program or that transform 15 endothelial cells. Such infectious disease include those bacterial infections that increase HIF-1 levels, HIV-Tat levels, antimicrobial peptides, levels, or those associated with tissue remodeling.

Infectious diseases, such as viral infections, can cause 20 excessive angiogenesis which is susceptible to treatment with agents of the invention. Examples of viruses that have been found in humans include, but are not limited to, Retroviridae (e.g. human immunodeficiency viruses, such as HIV-1 (also referred to as HDTV-III, LAVE or HTLV-III/LAV, or HIV-III; 25 and other isolates, such as HIV-LP; Picornaviridae (e.g. polio viruses, hepatitis A virus; enteroviruses, human Coxsackie viruses, rhinoviruses, echoviruses); Calciviridae (e.g. strains that cause gastroenteritis); Togaviridae (e.g. equine encephalitis viruses, rubella viruses); Flaviridae (e.g. dengue viruses, 30 encephalitis viruses, yellow fever viruses); Coronoviridae (e.g. coronaviruses); Rhabdoviridae (e.g. vesicular stomatitis viruses, rabies viruses); Filoviridae (e.g. ebola viruses); Paramyxoviridae (e.g. parainfluenza viruses, mumps virus, measles virus, respiratory syncytial virus); Orthomyxoviri- 35 dae (e.g. influenza viruses); Bungaviridae (e.g. Hantaan viruses, bunga viruses, phleboviruses and Nairo viruses); Arena viridae (hemorrhagic fever viruses); Reoviridae (e.g. reoviruses, orbiviurses and rotaviruses); Birnaviridae; Hepadnaviridae (Hepatitis B virus); Parvovirida (parvoviruses); 40 Papovaviridae (papilloma viruses, polyoma viruses); Adenoviridae (most adenoviruses); Herpesviridae (herpes simplex virus (HSV) 1 and 2, varicella zoster virus, cytomegalovirus (CMV), herpes virus; Poxyiridae (variola viruses, vaccinia viruses, pox viruses); and Iridoviridae (e.g. African swine 45 fever virus); and unclassified viruses (e.g. the agent of delta hepatitis (thought to be a defective satellite of hepatitis B virus), the agents of non-A, non-B hepatitis (class 1=internally transmitted; class 2=parenterally transmitted (i.e. Hepatitis C); Norwalk and related viruses, and astroviruses).

The present invention provides methods of treating diseases and/or disorders or symptoms thereof associated with excess or undesired angiogenesis, which comprise administering a therapeutically effective amount of a pharmaceutical composition comprising a compound of the formulae herein 55 to a subject (e.g., a mammal, such as a human). Thus, one embodiment is a method of treating a subject suffering from or susceptible to an angiogenesis-related disease or disorder or symptom thereof. The method includes the step of administering to the mammal a therapeutic amount of an amount of a compound herein sufficient to treat the disease or disorder or symptom thereof (e.g., to prevent or reduce angiogenesis) under conditions such that the disease or disorder is treated.

The methods herein include administering to the subject (including a subject identified as in need of such treatment) an 65 effective amount of a compound described herein (e.g., a peptide described herein, or mimetic, or analog thereof), or a

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composition described herein to produce such effect. Identifying a subject in need of such treatment can be in the judgment of a subject or a health care professional and can be subjective (e.g. opinion) or objective (e.g. measurable by a test or diagnostic method).

The therapeutic methods of the invention (which include prophylactic treatment) in general comprise administration of a therapeutically effective amount of the compounds herein, such as a compound of the formulae herein to a subject (e.g., animal, human) in need thereof, including a mammal, particularly a human. Such treatment will be suitably administered to subjects, particularly humans, suffering from, having, susceptible to, or at risk for a disease, disorder, or symptom thereof. Determination of those subjects "at risk" can be made by any objective or subjective determination by a diagnostic test or opinion of a subject or health care provider (e.g., genetic test, enzyme or protein marker, Marker (as defined herein), family history, and the like). The compounds herein may be also used in the treatment of any other disorders in which angiogenesis may be implicated.

In one embodiment, the invention provides a method of monitoring treatment progress. The method includes the step of determining a level of diagnostic marker (Marker) (e.g., any target delineated herein modulated by a compound herein, a protein or indicator thereof, etc.) or diagnostic measurement (e.g., screen, assay) in a subject suffering from or susceptible to a disorder or symptoms thereof associated with angiogenesis, in which the subject has been administered a therapeutic amount of a compound herein sufficient to treat the disease or symptoms thereof. The level of Marker determined in the method can be compared to known levels of Marker in either healthy normal controls or in other afflicted patients to establish the subject's disease status. In preferred embodiments, a second level of Marker in the subject is determined at a time point later than the determination of the first level, and the two levels are compared to monitor the course of disease or the efficacy of the therapy. In certain preferred embodiments, a pre-treatment level of Marker in the subject is determined prior to beginning treatment according to this invention; this pre-treatment level of Marker can then be compared to the level of Marker in the subject after the treatment commences, to determine the efficacy of the treatment.

#### Treatment of Neoplasia

The methods of the invention are particularly well suited for the treatment of neoplasias. By "neoplasia" is meant a disease that is caused by or results in inappropriately high levels of cell division, inappropriately low levels of apoptosis, or both. For example, cancer is an example of a proliferative disease. Examples of cancers include, without limitation, leukemias (e.g., acute leukemia, acute lymphocytic leukemia, acute myelocytic leukemia, acute myeloblastic leukemia, acute promyelocytic leukemia, acute myelomonocytic leukemia, acute monocytic leukemia, acute erythroleukemia, chronic leukemia, chronic myelocytic leukemia, chronic lymphocytic leukemia), polycythemia vera, lymphoma (Hodgkin's disease, non-Hodgkin's disease), Waldenstrom's macroglobulinemia, heavy chain disease, and solid tumors, such as sarcomas and carcinomas (e.g., fibrosarcoma, myxosarcoma, liposarcoma, chondrosarcoma, osteogenic sarcoma, chordoma, angiosarcoma, endotheliosarcoma, lymphangiosarcoma, lymphangioendotheliosarcoma, synovioma, mesothelioma, Ewing's tumor, leiomyosarcoma, rhabdomyosarcoma, colon carcinoma, pancreatic cancer, breast cancer, ovarian cancer, prostate cancer, squamous cell carcinoma, basal cell carcinoma, adenocarcinoma, sweat gland carcinoma, sebaceous gland carcinoma, papillary car-

cinoma, papillary adenocarcinomas, cystadenocarcinoma, medullary carcinoma, bronchogenic carcinoma, renal cell carcinoma, hepatoma, nile duct carcinoma, choriocarcinoma, seminoma, embryonal carcinoma, Wilm's tumor, cervical cancer, uterine cancer, testicular cancer, lung carcinoma, 5 small cell lung carcinoma, bladder carcinoma, epithelial carcinoma, glioma, astrocytoma, medulloblastoma, craniopharyngioma, ependymoma, pinealoma, hemangioblastoma, acoustic neuroma, oligodenroglioma, schwannoma, meningioma, melanoma, neuroblastoma, and retinoblastoma). 10 Lymphoproliferative disorders are also considered to be proliferative diseases.

#### Peptides of the Invention

The present invention utilizes powerful computational and bioinformatic approaches to identify therapeutic agents (e.g., 15 polypeptides, peptides, analogs, and fragments thereof) having anti-angiogenic activity. The amino acid sequences of such agents are provided herein. The Tables and Figures provide sequences of peptides of the invention, GenBank Accession Nos., and the amino acid positions of the 20 sequences. Amino acids are referred to herein by their commonly known one-letter symbols recommended by the IUPAC-IUB Biochemical Nomenclature Commission; they can also be referred to by their commonly known three letter symbols.

#### Angiogenesis Assays

The biological activity of therapeutic agents of the invention is characterized using any method for assaying angiogenic activity known in the art. In vitro angiogenesis assays have been described in detail in recent reviews (Akhtar et al., 30 Angiogenesis, 5:75-80, 2002; Auerbach et al., Cancer Metastasis Rev, 19:167-72, 2000; Auerbach et al., Clin Chem, 49:32-40, 2003; Staton et al., Int J Exp Pathol, 85:233-48, 2004; Vailhe et al., Lab Invest, 81:439-52, 2001). There are a number of different endothelial cell lineages that have been 35 used in angiogenesis assays: bovine aortic, bovine retinal, rat and mouse microvascular, human aortic, human bladder microvascular, human cardiac microvascular, human dermal microvascular, human lung microvascular and human umbilical vein endothelial cells. All of these endothelial cells are 40 capable of differentiating in vitro and forming capillary-like structures. This process occurs when the cells are cultured in a monolayer of extracellular matrix components, such as the Matrigel (extracellular matrix material similar to basement membrane), type I collagen, fibronectin or laminin. An endot- 45 helial cell lineage that is commonly used for testing the angiogenic response is the human umbilical vein endothelial cells (HUVECs). The National Cancer Institute (NCI) has issued guidelines for testing the anti-angiogenic efficacy of novel agents; they include proliferation, migration and tube forma- 50 tion assays using HUVECs.

Initially the anti-angiogenic effect of selected standard agents is assessed as a positive control by adding them into the wells containing cultured endothelial cells. Such standard anti-angiogenic agents include the fumigillin analog TNP- 470 that is available by request from NCI. The standard cell culture medium is usually used as a negative control. The experiments described below are repeated several times as required to obtain statistically significant and reproducible results. Once the platform is calibrated and tested for the 60 known agents, the novel inhibitors are tested.

#### Cell Proliferation Assay

In these assays anti-angiogenic agents are tested for their ability to alter endothelial cell proliferation. A reduction in endothelial cell proliferation identifies an agent that inhibits 65 angiogenesis. The viability and metabolic activity of the cells is measured in the presence of the anti-angiogenic peptides at

different concentrations and various time steps. In one example, a cell proliferation reagent, MTT, is used in a substrate/assay that measures the metabolic activity of viable cells. The assay is based on the reduction of the vellow tetrazolium salt, MTT, by viable, metabolically active cells to form the insoluble purple formazan crystals, which are solubilized by the addition of a detergent. MTT is a colorimetric, non-radioactive assay that can be performed in a microplate. It is suitable for measuring cell proliferation, cell viability or cytotoxicity. The procedure involves three steps. First, the cells are cultured in a multi-well plate and then incubated with the yellow MTT for approximately 2 to 4 hours. During this incubation period, viable cells convert, in their mitochondria, the yellow MTT to the purple formazan crystals. The second step involves the solubilization of the crystals. A detergent solution is added to lyse the cells and solubilize the colored crystals. The final step of the assay involves quantifying changes in proliferation by measuring the changes in the color after lysing the cells. The samples are read using an ELISA plate reader at a wavelength of 570 nm. The amount of color produced is directly proportional to the number of viable cells present in a particular well. Other proliferation assays include WST-1, XTT, Trypan Blue, Alamar Blue and BrdU. In contrast to the MTT assay, in the WST-1 assay the formazan crystals do not need to be solubilized by the addition of a detergent; they are soluble to the cell medium.

In another example, cell proliferation is assayed by quantitating bromodeoxyuridine (BrdU) incorporation into the newly synthesized DNA of replicating cells. The assay is a cellular immunoassay that uses a mouse monoclonal antibody directed against BrdU. The procedure involves four steps. First, the cells are cultured in a microtiterplate and pulse-labeled with BrdU. Only proliferating cells incorporate BrdU into their DNA. The cells are then fixed in a denaturing solution. The genomic DNA is denatured, exposing the incorporated BrdU to immunodetection. The BrdU label is located in the DNA with a peroxidase-conjugated anti-BrdU antibody. The antibody is quantitated using a peroxidase substrate. To test anti-proliferative effects of the selected peptides, the endothelial cells are incubated in the presence of varying amounts of the peptides for different time intervals. After labeling of the cells with BrdU the cell proliferation reagent WST-1 is added, and the cells are reincubated. The formazan product is quantified at 450 nm with an absorbance reader. Subsequently, BrdU incorporation is determined using the colorimetric cell proliferation ELISA, BrdU. The results of this assay indicate the effects of the anti-angiogenic peptides either on DNA synthesis (anti-proliferative) or the metabolic activity (pro-apoptotic) of the cell. Kits for implementing these techniques are commercially available.

Preferably, an agent of the invention reduces cell proliferation by at least about 5%, 10%, 20% or 25%. More preferably, cell proliferation is reduced by at least 50%, 75%, or even by 100%

#### Cell Apoptosis and Cell Cycle Assay

Agents having anti-angiogenic activity can also be identified in assay that measures the effect of a candidate agent on cell proliferation and survival using a mitogenic assay (incorporation of thymidine, or 5-bromodeoxyuridine) that measures alterations in cell number (direct counts or indirect colorimetric evaluation). Agents that reduce cell proliferation, cell survival, or that increase cell death are identified as having anti-angiogenic activity. Cell death by apoptosis can be measured using a TUNEL assay or by analyzing the expression of apoptosis markers, such as the caspases and annexin V (Fennell et al., J Biomol Screen, 11:296-302, 2006;

Loo and Rillema, *Methods Cell Biol*, 57:251-64, 1998; Otsuki et al., *Prog Histochem Cytochem*, 38:275-339, 2003).

A number of methods have been developed to study apoptosis in cell populations. Apoptosis is a form of cell death that is characterized by cleavage of the genomic DNA into 5 discrete fragments prior to membrane disintegration. Because DNA cleavage is a hallmark for apoptosis, assays that measure prelytic DNA fragmentation are especially attractive for the determination of apoptotic cell death. DNA fragments obtained from cell populations are assayed on agarose gels to identify the presence of absence of "DNA ladders" or bands of 180 bp multiples, which form the rungs of the ladders, or by quantifying the presence of histone complexed DNA fragments by ELISA.

Other indicators of apoptosis include assaying for the presence caspases that are involved in the early stages of apoptosis. The appearance of caspases sets off a cascade of events that disable a multitude of cell functions. Caspase activation can be analyzed in vitro by utilizing an enzymatic assay. Activity of a specific caspase, for instance caspase 3, can be 20 determined in cellular lysates by capturing of the caspase and measuring proteolytic cleavage of a suitable substrate that is sensitive to the specific protease (Fennell et al., *J Biomol Screen*, 11:296-302, 2006; Loo and Rillema, *Methods Cell Biol*, 57:251-64, 1998; Otsuki et al., *Prog Histochem* 25 *Cytochem*, 38:275-339, 2003). Agents that increase caspase activity or DNA fragmentation in endothelial cells are identified as useful in the methods of the invention.

In addition to in vitro techniques, apoptosis can be measured using flow cytometry. One of the simplest methods is to 30 use propidium iodide (PI) to stain the DNA and look for sub-diploid cells (Fennell et al., *J Biomol Screen*, 11:296-302, 2006; Loo and Rillema, *Methods Cell Biol*, 57:251-64, 1998; Otsuki et al., *Prog Histochem Cytochem*, 38:275-339, 2003).

The most commonly used dye for DNA content/cell cycle analysis is propidium iodide (PI). PI intercalates into the major groove of double-stranded DNA and produces a highly fluorescent adduct that can be excited at 488 nm with a broad emission centered around 600 nm. Since PI can also bind to double-stranded RNA, it is necessary to treat the cells with 40 RNase for optimal DNA resolution. Other flow cytometric-based methods include the TUNEL assay, which measures DNA strand breaks and Annexin V binding, which detects relocation of membrane phosphatidyl serine from the intracellular surface to the extracellular surface.

#### Cell Migration and Invasion Assay

Another anti-angiogenic activity is the ability to reduce endothelial cell migration towards an attractant that is present in a chemotactic gradient, such as a growth factor gradient. Endothelial cell motility or migration can be assessed using 50 the Boyden chamber technique (Auerbach et al., Cancer Metastasis Rev, 19:167-72, 2000; Auerbach et al., Clin Chem, 49:32-40, 2003; Taraboletti and Giavazzi, Eur J Cancer, 40:881-9, 2004). In one example, a Boyden chamber assay is used to test endothelial cell migration from one side of the 55 chamber in the presence of an activator. In brief, the lower compartment of the Boyden chamber is separated from the upper (containing the endothelial cells) by a matrix-coated polycarbonate filter with pores small enough to allow only the active passage of the cells (3-8 µm pore size). The matrix may 60 include, for example, extracellular matrix proteins, such as collagen, laminin and fibronectin. Activators include but are not limited to growth factors, such as vascular endothelial growth factor and fibroblast growth factor-2 or conditioned medium (e.g. from tumor cells or NIH-3T3 fibroblasts). 65 Migration typically occurs rapidly typically within 4-20 hours cells have migrated through the filter. The number of

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migrating cells is quantified using a cell-permeable fluorescent dye in the presence or absence of an inhibitor; it can also be quantified by any means of cell counting. A fluorescence plate reader is used to quantify the migrating cells by measuring the amount of fluorescence and directly correlating it to cell number. A decrease in cell migration identifies a peptide that inhibits angiogenesis. Preferably, cell migration or motility is reduced by at least about 5%, 10%, 20% or 25%. More preferably, cell migration or motility is reduced by at least about 50%, 75%, or even by 100%.

In other embodiments, anti-angiogenic agents of the invention alter the invasiveness of an endothelial cell, for example, by reducing the ability of an endothelial cell to degrade an extracellular matrix component. In one example, an anti-angiogenic inhibitor acts by reducing the proteolytic activity of a matrix metalloproteinase. Methods for assaying protease activity are known in the art. Quantification of the matrix metalloproteinase activity can be accomplished using a zymographic or gelatinase activity assay (Frederiks and Mook, *J Histochem Cytochem*. 52:711-22, 2004). Preferably, protease activity is reduced by at least about 5%, 10%, 20% or 25%. More preferably, protease activity is reduced by at least about 50%, 75%, or even by 100%.

In another example, the invasive activity of an endothelial cell is measured using a Boyden chamber invasion assay or by measuring phagokinetic tracks. The invasion assay is essentially as described above for the Boyden motility assay, except that the filter is coated with a layer of a matrix several microns thick, usually Matrigel or other basement membrane extracts, which the cells must degrade before migrating through the filter (Auerbach et al., *Cancer Metastasis Rev*, 19:167-72, 2000; Auerbach et al., *Clin Chem*, 49:32-40, 2003; Taraboletti and Giavazzi, *Eur J Cancer*, 40:881-9, 2004). Compounds that reduce extracellular matrix degradation or endothelial cell invasiveness are identified as useful in the methods of the invention.

#### Tube Formation Assay

Another method of identifying an agent having anti-angiogenic activity involves measuring the agent's ability to reduce or disrupt capillary tube formation. Various types of endothelial cells (e.g., HUVECs, HMVECs (human microvascular endothelial cells)) form tubes when cultured in wells uniformly coated with Matrigel, an extracellular matrix protein, or other substrates. Therefore the assay characterizes endothelial cell differentiation. The endothelial cells are cultured in the presence or the absence of a candidate agent. The agent may be added to the culture media or may be present or applied to the gel. Typically, the effect on tube formation is measured by incubating the cells for a period of time (e.g., one to four days) at 37° C. in 5% CO<sub>2</sub> atmosphere. Kits for implementing these techniques are commercially available.

The output of the experiments are images of capillary networks formed. A common metric used for the morphological characteristics of a capillary network is the angiogenic index. This index is calculated as the ratio of the total length of the connected tubes over the total monitored surface of the well. The change of the angiogenic index as a function of the concentration of the anti-angiogenic peptide will be the determinant for the effectiveness of the tested novel angiogenesis inhibitors.

#### Aortic Ring Assay

The aortic ring assay integrates the advantages of both in vivo and in vitro systems. It is a useful assay to test angiogenic factors or inhibitors in a controlled environment. More importantly, it recapitulates all of the necessary steps involved in angiogenesis (Staton et al., *Int J Exp Pathol*, 85:233-48, 2004).

In this quantitative method of studying angiogenesis, ring segments of aortas from various animals such as rats and mice are embedded in a three-dimensional matrix composed of fibrin or collagen, and cultured in a defined medium devoid of serum and growth factors. Microvessels sprout spontane- 5 ously from the surface of the aortic rings. This angiogenic process is mediated by endogenous growth factors produced from the aorta or can be assisted by applying exogenously specific concentrations of growth factors. The embedded aortas are incubated for 10-12 days and after the incubation 10 period the newly formed vessels are quantified. Microvessels can be counted manually or quantified using computer-assisted image analysis. Test agents can be added to the culture medium to assay for angiogenic or anti-angiogenic activity. Also aortas from animals with different genetic background 15 (e.g., knockout mice) can be used in order to assess specific mechanisms of the effect of the anti-angiogenic peptides on the neovessel formation process.

#### In Vivo Angiogenesis Assays

A recent review identified over 70 disease conditions that 20 involve angiogenesis, about half of those characterized by abnormal or excessive angiogenesis or lymphangiogenesis (Carmeliet, *Nature*, 438:932-6, 2005). Agents identified as having anti-angiogenic activity are optionally tested in in vivo assays using animal models that exhibit abnormal or excessive angiogenesis or lymphangiogenesis.

#### Matrigel Plug Assay

In one in vivo approach, a candidate agent of the invention is tested for anti-angiogenic activity by implanting a polymer matrix subcutaneously in an animal and assaying the matrix 30 for signs of neovascularization. In one embodiment, a Matrigel plug or a similar substrate containing tumor cells and an anti-angiogenic factor is used to study in vivo angiogenesis (Auerbach et al., Cancer Metastasis Rev, 19:167-72, 2000; Staton et al., Int J Exp Pathol, 85:233-48, 2004). Matrigel is a 35 liquid at 4° C., but forms a solid gel at 37° C. A candidate agent is suspended together with an attractant, such as a growth factor, in the gel. The Matrigel is then injected subcutaneously where it forms a solid plug allowing for the prolonged local release of pro- or anti-angiogenic agents 40 present in the gel. The plug is subsequently removed and neovascularization is assessed by any standard methods, including but not limited to, identifying the presence of endothelial cells or endothelial cell tubules in the plug using microscopy. In some embodiments, this approach is com- 45 bined with an immuno-histological identification of endothelium specific proteins (e.g., CD-31/34 or integrins) on the newly formed vessels.

The Matrigel plug assay can be applied for testing the efficacy of the novel anti-angiogenic peptides identified 50 herein. In one example, Matrigel is mixed with heparin (usually 20 U/ml) and a vascular endothelial growth factor at about 50 ng/ml in the presence or absence of a candidate peptide, which is supplied at a variety of concentrations (e.g., at the IC<sub>50</sub>). A control animal receives the gel without the 55 anti-angiogenic fragment. The Matrigel is injected into the mice subcutaneously and after one week the animals are sacrificed. The Matrigel plugs are then removed and fixed with 4% paraformaldehyde. The plugs are then embedded in paraffin, sectioned and stained with hematoxylin and eosin. 60 The number of blood vessels as well as any other angiogenic indexes are estimated.

#### Directed In Vivo Angiogenesis Assay (DIVAA)

Directed in vivo angiogenesis assay (DIVAA) is a reproducible and quantitative in vivo method of assaying angio-65 genesis. It involves the preparation of silicon cylinders that are closed on one side filled with some type of extracellular

matrix (for example Matrigel) with or without premixed angiogenic factors (Guedez et al., Am J Pathol, 162:1431-9, 2003) to form an angioreactor. The angioreactors are then implanted subcutaneously in mice. Vascular endothelial cells migrate into the extracellular matrix and form vessels in the angioreactor. As early as nine days post-implantation, there are enough cells present in the angioreactor to assay the effect of an angiogenic modulating factors. A candidate agent may be included in the matrix together with the angiogenic factors. The design of the angioreactor provides a standardized platform for reproducible and quantifiable in vivo angiogenesis assays.

Advantageously, the angioreactor prevents assay errors due to absorption of the basement membrane extract or the diffusion of the anti-angiogenic agent into the surrounding tissue; may be carried out using only a fraction of the materials required in the plug assay described above; and up to four angioreactors may be implanted in a single animal (e.g., mouse), providing more data for analysis. Vascularization response can be measured by intravenous injection of fluorescein isothiocyanate (FITC)-dextran before the recovery of the angioreactor, followed by spectrofluorimetry. Alternatively, to obtain a quantitative assessment of the angiogenic invasion, the content of the angioreactors, can be removed and the endothelial cells stained using FITC-Lectin. Fluorescence of the FITC-Lectin solution can be quantitated by measuring the fluorescence at 485 nm excitation and 510 nm emission using a fluorescence plate reader e.g., Victor 3V (Perkin Elmer). The intensity of the signal is directly proportional to the number of endothelial cells that are present in the angioreactors. The technique allows dose response analysis and identification of effective doses of angiogenesis-modulating factors in vivo.

#### Chorioallantoic Membrane Assay

The chorioallantoic membrane assay (CAM) is widely used as an angiogenesis assay Auerbach et al., Cancer Metastasis Rev 19:167-172, 2000; Staton et al., Int J Exp Pathol 85: 233-248, 2004; D'Amato, In: Voest, E. E., and D'Amore, P. A. (eds). Tumor Angiogenesis Microcirculation, 2001, Marcel Dekker, New York-Base1). In one embodiment, the chorioallantoic membrane of a 7-9 day old chick embryos is exposed by making a window in the egg shell. A candidate agent is provided in a formulation that provides for its extended release (e.g., in a slow-release polymer pellets, absorbed on a gelatin sponge, or air-dried onto a plastic disc). The candidate agent formulation is implanted onto the chorioallantoic membrane through a window in the shell. The window is sealed and the egg is re-incubated. The lack of mature immune system in the 7 day old chick embryos allows the study of angiogenesis without any immunological interference. In the modified version of the in ovo assay, the entire egg content is transferred to a plastic culture dish. After 3-6 days of incubation the testing agents are applied and angiogenesis is monitored using various angiogenesis indexes.

In the case of testing the angiostatic peptides, polymer pellets can be loaded both with the growth factors and the anti-angiogenic fragments and be implanted in the chorioal-lantoic membrane. The modified version of the assay allows the application of a candidate agent using different strategies to identify effective therapeutic regimens. For example, a candidate agent is applied in a single bolus at a particular concentration; at different time points at lower concentrations; or in different formulations that provide for the extended release of an agent. This provides for the temporal control of candidate agent release and the delineation of temporal variations in drug administration on the angiostatic activity of the candidate agents.

Ocular Angiogenesis Models

Corneal Micropocket:

The cornea is an avascular site and presumably any vessels penetrating from the limbus into the cornea stroma can be identified as newly formed. In this assay a pocket is created in the cornea stroma of the animal. An angiogenic response is usually initiated by implantation of a slow release pellet or polymer containing growth factors (Auerbach et al., *Cancer Metastasis Rev.* 19:167-72, 2000; Auerbach et al., *Clin Chem.* 49:32-40, 2003; D'Amato, *Tumor Angiogenesis and Microcirculation*, 103-110, 2001; Staton et al., *Int J Exp Pathol.* 85:233-48, 2004).

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In order to test an angiogenesis inhibitor, the effect of a candidate agent on an angiogenic response in the cornea is assayed after the implantation of a pellet comprising an angiogenic agent in combination with a candidate inhibitor in the cornea pockets. Also the efficacy of an anti-angiogenic agent can be evaluated using the mouse model of ocular ischemic retinopathy to quantitatively assess anti-angiogenic effects on retinal neovascularization. In addition, a mouse model of laser induced choroidal neovascularization can be used in order to quantitatively assess the anti-angiogenic effects of candidate agents on choroidal neovascularization.

The tested peptides can be administered with a bolus injection 25 or any other scheduled administration.

Mouse Model of Choroidal Neovascularization (CNV):

Laser photocoagulation is used on normal mice to rupture Bruch's membrane at three locations in each eye (e.g., To be et al., Am J Pathol 153:1641-1646, 1998); this procedure leads to neovascularization arising from the choroidal circulation. On the day of laser treatment, the mice are injected intravitreously with the peptide being evaluated. The injections are repeated a week later. One eye is injected with peptide, the contralateral eye receives the vehicle or scrambled peptide as control. Two weeks following laser treatment the mice are sacrificed and quantitative assessment of choroidal neovascularization is performed. The eyes are removed and fixed overnight in phosphate-buffered formalin. 40 The cornea and lens are removed and the entire retina is dissected from the eyecup. Radial cuts are made from the edge to the equator and the eyecup is flat mounted with the sclera facing down. Flat mounts are examined by fluorescence microscopy. The area of the CNV lesions in the peptide 45 injected eyes are compared to the area of neovascularization of CNV in the paired vehicle injected eyes.

Mouse Model of Ischemic Retinopathy:

Seven-day-old (P7) mice and their mothers are placed in an airtight incubator and exposed to an atmosphere of 75% oxy-50 gen for 5 day (Smith et al., Invest Ophthalmol Vis Sci. 35:101-111, 1994). The incubator temperature is maintained at 23° C., and oxygen is continuously monitored with an oxygen controller. At P12 the litters are returned to room air. One day following removal from oxygen and return to room air intra- 55 vitreous injection of peptide into the right eye of each pup and vehicle into the left is carried out. On P17 pups are euthanized, and the eyes are rapidly removed, positioned and frozen in an embedding compound. Ocular sections are then stained with Griffonia Simplicifolia lectin that labels vascular 60 endothelial cells. Histopathological sections demonstrating the presence, extent and location of normal and abnormal blood vessels are then analyzed following preparation of a standardized series of sections in each eye. The area of retinal neovascularization in the peptide injected eye is compared to 65 the area of retinal neovascularization in the vehicle injected

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Chamber Assays

Other methods for studying the effect of a candidate agent in vivo on chronic angiogenesis involve the use of an implanted transparent chamber. The chamber is implanted in an accessible site (e.g., the rabbit ear, the dorsal skinfold and the cranial window chamber (Auerbach et al., Clin Chem, 49:32-40, 2003; Staton et al., Int J Exp Pathol, 85:233-48, 2004). In each of these systems a piece of skin (the ear or skinfold chamber) or part of the skull (cranial chamber) is removed from an anesthetized animal. Tumor cells or a pellet containing an angiogenesis stimulus is then placed on the exposed surface and covered by a glass. The animals are allowed to recover, and angiogenesis is subsequently monitored. The models allow for the continuous measurement of various angiogenesis as well as tissue parameters, such as pH or blood flow. Similarly to the corneal pocket assay, the angiostatic agents are administered orally, locally, or systemically using a predefined drug administration schedule. Agents that reduce angiogenesis in a chamber assay are identified as useful in the methods of the invention.

Tumor Models

Many different in vivo models have been developed to test the activity of potential anti-angiogenic or anti-cancer treatments, specifically on tumor vasculature. Tumors are implanted and can be grown syngeneically; i.e., subcutaneously, orthotopically in a tissue of origin, or as xenografts in immunodeficient mice (Auerbach et al., *Clin Chem*, 49:32-40, 2003; Staton et al., *Int J Exp Pathol*, 85:233-48, 2004). Any number of histological analyses may be used to examine the effect of a candidate agent on a blood vessel supplying the tumor. In one embodiment, the blood vessel density of a newly formed vasculature in the tumor is monitored; in another embodiment, the vascular architecture is monitored, for example, by counting the number of vascular branches per vessel unit length. In another embodiment, blood flow through the vasculature is measured.

The tumor models provide a variety of different conditions that can be analyzed to assay the efficacy of a candidate anti-angiogenic agent. For example, the effects of a candidate agent on the stability of a well vascularized vs. a poorly vascularized tumor can be assayed; the effect of a candidate agent on tumors of different origin, for example prostate and breast cancer, renal cell carcinoma, and including those of vascular origin such as the chemically induced hemangiosarcomas and Kaposi's sarcomas, can be analyzed. The study of in vivo tumor models provide the closest approximation of human tumor angiogenesis. Moreover, such models provide the opportunity to study the pharmacokinetics of the candidate drug as well as its efficacy simultaneously in a large scale model and under different administration carriers and strategies.

Anti-Angiogenic Peptides and Analogs

The invention is not limited to conventional therapeutic peptides having anti-angiogenic activity, but comprises a variety of modified peptides having properties that enhance their biodistribution, selectivity, or half-life. In particular, the invention provides peptides that are modified in ways that enhance their ability to inhibit angiogenesis in a cell, tissue, or organ in a subject in need thereof.

The invention provides methods for optimizing a transcription factor or protein transduction domain amino acid sequence or nucleic acid sequence by producing an alteration in the sequence. Such alterations may include certain mutations, deletions, insertions, or post-translational modifications. The invention further includes analogs of any naturally-occurring polypeptide of the invention. Analogs can differ from a naturally-occurring polypeptide of the invention by

amino acid sequence differences, by post-translational modifications, or by both. Analogs of the invention will generally exhibit at least 85%, more preferably 90%, and most preferably 95% or even 99% identity with all or part of a naturallyoccurring amino, acid sequence of the invention. The length 5 of sequence comparison is at least about 5, 10, 15 or 20 amino acid residues, at least about 25, 50, or 75 amino acid residues, or at least about 100 amino acid residues. Again, in an exemplary approach to determining the degree of identity, a BLAST program may be used, with a probability score 10 between  $e^{-3}$  and  $e^{-100}$  indicating a closely related sequence. Modifications include in vivo and in vitro chemical derivatization of polypeptides, e.g., acetylation, carboxylation, phosphorylation, or glycosylation; such modifications may occur during polypeptide synthesis or processing or following treat- 15 ment with isolated modifying enzymes. Analogs can also differ from the naturally-occurring polypeptides of the invention by alterations in primary sequence. These include genetic variants, both natural and induced (for example, resulting from random mutagenesis by irradiation or exposure to 20 ethanemethylsulfate or by site-specific mutagenesis as described in Sambrook, Fritsch and Maniatis, Molecular Cloning: A Laboratory Manual (2d ed.), CSH Press, 1989, or Ausubel et al., supra). Also included are cyclized peptides, molecules, and analogs which contain residues other than 25 L-amino acids, e.g., D-amino acids or non-naturally occurring or synthetic amino acids, e.g.,  $\beta$  or  $\gamma$  amino acids.

The term "amino acid" refers to naturally occurring and synthetic amino acids, as well as amino acid analogs and amino acid mimetics that function in a manner similar to the 30 naturally occurring amino acids. Naturally occurring amino acids are those encoded by the genetic code, as well as those amino acids that are later modified, for example, hydroxyproline, gamma-carboxyglutamate, and O-phosphoserine, phosphothreonine. "Amino acid analogs" refer to compounds that 35 have the same basic chemical structure as a naturally occurring amino acid, i.e., a carbon that is bound to a hydrogen, a carboxyl group, an amino group, and an R group, for example, homoserine, norleucine, methionine sulfoxide, methionine methyl sulfonium. Such analogs have modified R 40 groups (for example, norleucine) or modified peptide backbones, but retain the same basic chemical structure as a naturally occurring amino acid. "Amino acid mimetics" refers to chemical compounds that have a structure that is different from the general chemical structure of an amino acid, but that 45 function in a manner similar to a naturally occurring amino acid. Amino acids and analogs are well known in the art. Amino acids may be referred to herein by either their commonly known three letter symbols or by the one-letter symbols recommended by the IUPAC-IUB Biochemical Nomen- 50 clature Commission. Nucleotides, likewise, may be referred to by their commonly accepted single-letter codes.

"Conservatively modified variants" apply to both amino acid and nucleic acid sequences. With respect to particular nucleic acid sequences, conservatively modified variants 55 refers to those nucleic acids which encode identical or similar amino acid sequences and include degenerate sequences. For example, the codons GCA, GCC, GCG and GCU all encode alanine. Thus, at every amino acid position where an alanine is specified, any of these codons can be used interchangeably 60 in constructing a corresponding nucleotide sequence. The resulting nucleic acid variants are conservatively modified variants, since they encode the same protein (assuming that is the only alternation in the sequence). One skilled in the art recognizes that each codon in a nucleic acid, except for AUG 65 (sole codon for methionine) and UGG (tryptophan), can be modified conservatively to yield a functionally-identical pep-

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tide or protein molecule. As to amino acid sequences, one skilled in the art will recognize that substitutions, deletions, or additions to a polypeptide or protein sequence which alter, add or delete a single amino acid or a small number (typically less than about ten) of amino acids is a "conservatively modified variant" where the alteration results in the substitution of an amino acid with a chemically similar amino acid. Conservative substitutions are well known in the art and include, for example, the changes of alanine to serine; arginine to lysine; asparigine to glutamine or histidine; aspartate to glutamate; cysteine to serine; glutamine to asparigine; glutamate to aspartate; glycine to proline; histidine to asparigine or glutamine; isoleucine to leucine or valine; leucine to valine or isoleucine; lysine to arginine, glutamine, or glutamate; methionine to leucine or isoleucine; phenylalanine to tyrosine, leucine or methionine; serine to threonine; threonine to serine; tryptophan to tyrosine; tyrosine to tryptophan or phenylalanine; valine to isoleucine or leucine. Other conservative and semi-conservative substitutions are known in the art and can be employed in practice of the present inven-

The terms "protein", "peptide" and "polypeptide" are used herein to describe any chain of amino acids, regardless of length or post-translational modification (for example, glycosylation or phosphorylation). Thus, the terms can be used interchangeably herein to refer to a polymer of amino acid residues. The terms also apply to amino acid polymers in which one or more amino acid residue is an artificial chemical mimetic of a corresponding naturally occurring amino acid. Thus, the term "polypeptide" includes full-length, naturally occurring proteins as well as recombinantly or synthetically produced polypeptides that correspond to a full-length naturally occurring protein or to particular domains or portions of a naturally occurring protein. The term also encompasses mature proteins which have an added amino-terminal methionine to facilitate expression in prokaryotic cells.

The polypeptides and peptides of the invention can be chemically synthesized or synthesized by recombinant DNA methods; or, they can be purified from tissues in which they are naturally expressed, according to standard biochemical methods of purification. Also included in the invention are "functional polypeptides," which possess one or more of the biological functions or activities of a protein or polypeptide of the invention. These functions or activities include the ability to inhibit angiogenesis (e.g., by reducing endothelial cell proliferation, migration, survival, or tube formation). The functional polypeptides may contain a primary amino acid sequence that has been modified from that considered to be the standard sequence of a peptide described herein. Preferably these modifications are conservative amino acid substitutions, as described herein.

In addition to full-length polypeptides, the invention also includes fragments of any one of the polypeptides of the invention. As used herein, the term "a fragment" means at least 5, 10, 13, or 15 amino acids. In other embodiments a fragment is at least 20 contiguous amino acids, at least 21, 22, 23, 24, or 25 contiguous amino acids, or at least 30, 35, 40, or 50 contiguous amino acids, and in other embodiments at least 60 to 80 or more contiguous amino acids. Fragments of the invention can be generated by methods known to those skilled in the art or may result from normal protein processing (e.g., removal of amino acids from the nascent polypeptide that are not required for biological activity or removal of amino acids by alternative mRNA splicing or alternative protein processing events).

Non-protein transcription factor/protein transduction domain fusion analogs have a chemical structure designed to

mimic the fusion proteins functional activity. Such analogs are administered according to methods of the invention. Fusion protein analogs may exceed the physiological activity of the original fusion polypeptide. Methods of analog design are well known in the art, and synthesis of analogs can be carried out according to such methods by modifying the chemical structures such that the resultant analogs increase the reprogramming or regenerative activity of a reference transcription factor/protein transduction domain fusion polypeptide. These chemical modifications include, but are not limited to, substituting alternative R groups and varying the degree of saturation at specific carbon atoms of a reference fusion polypeptide. Preferably, the fusion protein analogs are relatively resistant to in vivo degradation, resulting in 15 a more prolonged therapeutic effect upon administration. Assays for measuring functional activity include, but are not limited to, those described in the Examples below.

Iterative design approaches (DeFreest et al., *J Pept Res*, 20 63:409-19, 2004) offer unique opportunities to optimize the structure and function of the candidate anti-angiogenic peptides. During iterative design an initial set of amino acids is substituted and the effect of the resulting agent on angiogenesis is assayed. The exploration of the structure-function relationships, but most importantly the conservation of the biophysical and biochemical characteristics of the peptides, during the iterative design and synthesis, is expected to contribute to the optimization of the anti-angiogenic activity. To determine which residues are essential to the bioactivity of the predicted peptide a series of analogs is prepared and evaluated.

Peptide-Design Approaches

In order to assess the types of substitutions within the amino acid sequence of the candidate peptide one can initially use computational methods. The most straightforward method for deciphering the importance of each amino acid is to investigate the conservation of these amino acids at multiple orthologues (same locus in different organisms). Amino acids that are conserved among different organisms are iden- 40 tified as functionally significant. From a biophysical point of view electrostatic interactions and hydrophobic partitioning act in concert to promote the interactions of the peptides with their receptors. In this sense, any point substitution should comply with the conservation of the net charge and hydro- 45 phobicity of the agent (DeFreest et al., JPept Res, 63:409-19, 2004). Phage display technology can also be used for performing random substitutions at expressed peptides of 20-25 amino acids length (Scott and Smith, Science, 249:386-90, 1990). In each of the cases the resultant peptide is tested for its 50 effect on angiogenesis using any of the assays described

Design optimization of the activity of the predicted peptides can also be performed by altering specific structural characteristics of the agents. For example, it has been shown 55 (DeFreest et al., *J Pept Res*, 63:409-19, 2004) that head-to-tail cyclization of the molecules confers an active dose range broader than the linear form of the molecules, and the peptide stability and shelf life are not compromised. The head-to-tail conjunction can occur either by a disulfide bond or by a 60 peptide bond formation. The use of a peptide bond may be advantageous for purposes of shelf life, and elimination of dimers, trimers, and higher-order aggregates formation that can sometimes develop when peptides are stored or used in conditions where the redox state cannot be fully controlled. 65 The cyclization approaches are discussed in the following section.

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Cyclization of Linear Peptides

Cyclization of peptides has been shown to be a useful approach to developing diagnostically and therapeutically useful peptidic and peptidomimetic agents. Cyclization of peptides reduces the conformational freedom of these flexible, linear molecules, and often results in higher receptor binding affinities by reducing unfavorable entropic effects. Because of the more constrained structural framework, these agents are more selective in their affinity to specific receptor cavities. By the same reasoning, structurally constrained cyclic peptides confer greater stability against the action of proteolytic enzymes.

Methods for cyclization can be classified into the so called "backbone to backbone" cyclization by the formation of the amide bond between the N-terminal and the C-terminal amino acid residues, and cyclizations involving the side chains of individual amino acids (Li and Roller, Curr Top Med Chem, 2:325-41, 2002). Although many novel approaches have been developed to accomplish the head-totail cyclization of linear peptides and peptidomimetics, the most commonly used method is still the solution phase macro-cyclization using peptide coupling reagents. The results of the peptide cyclization are mainly influenced by the conformation of the linear peptide precursors in solution. Synthesis design is affected by the strategy of the ring disconnection, and the rational selection of peptide coupling reagents. A reasonable ring disconnection will significantly facilitate the peptide macro-cyclization reaction, while a poor selection of cyclization site may result in slow reaction speed and low yield accompanied by various side reactions such as racemization, dimerization, and oligomerization.

Cyclization involving the side chains of individual amino acids includes the formation of disulfide bridges between omega-thio amino acid residues (cysteine, homocysteine), the formation of lactam bridges between glutamic/aspartic acid and lysine residues, the formation of lactone or thiolactone bridges between amino acid residues containing carboxyl, hydroxyl or mercapto functional groups, and the formation of thio-ether or ether bridges between the amino acids containing hydroxyl or mercapto functional groups.

Recombinant Polypeptide Expression

The invention provides therapeutic peptides that are most commonly generated by routine methods for peptide synthesis. Such methods are known in the art and are described herein. If an alternative approach is desired, the peptides are expressed recombinantly, either alone, or as part of a larger fusion protein that includes an anti-angiogenic peptide operably linked to a polypeptide that facilitates expression. If desired, the peptide can subsequently be cleaved (e.g., enzymatically) from the fusion protein. Where the fusion protein does not interfere with the anti-angiogenic activity of the peptide such cleavage may not be necessary or even desirable. When the therapeutic peptide or fusion protein comprising the peptide contacts an endothelial cell, tissue, or organ comprising such a cell it reduces angiogenesis. Recombinant polypeptides of the invention are produced using virtually any method known to the skilled artisan. Typically, recombinant polypeptides are produced by transformation of a suitable host cell with all or part of a polypeptide-encoding nucleic acid molecule or fragment thereof in a suitable expression vehicle.

Those skilled in the field of molecular biology will understand that any of a wide variety of expression systems may be used to provide the recombinant protein. The precise host cell used is not critical to the invention. A polypeptide of the invention may be produced in a prokaryotic host (e.g., *E. coli*) or in a eukaryotic host (e.g., *Saccharomyces cerevisiae*, insect

cells, e.g., Sf21 cells, or mammalian cells, e.g., NIH 3T3, HeLa, or preferably COS cells). Such cells are available from a wide range of sources (e.g., the American Type Culture Collection, Rockland, Md.; also, see, e.g., Ausubel et al., Current Protocol in Molecular Biology, New York: John 5 Wiley and Sons, 1997). The method of transformation or transfection and the choice of expression vehicle will depend on the host system selected. Transformation and transfection methods are described, e.g., in Ausubel et al. (supra); expression vehicles may be chosen from those provided, e.g., in 10 Cloning Vectors: A Laboratory Manual (P. H. Pouwels et al., 1985, Supp. 1987).

A variety of expression systems exist for the production of the polypeptides of the invention. Expression vectors useful for producing such polypeptides include, without limitation, 15 chromosomal, episomal, and virus-derived vectors, e.g., vectors derived from bacterial plasmids, from bacteriophage, from transposons, from yeast episomes, from insertion elements, from yeast chromosomal elements, from viruses such as baculoviruses, papova viruses, such as SV40, vaccinia 20 viruses, adenoviruses, fowl pox viruses, pseudorabies viruses and retroviruses, and vectors derived from combinations thereof.

One particular bacterial expression system for polypeptide production is the E. coli pET expression system (e.g., pET- 25 28) (Novagen, Inc., Madison, Wis.). According to this expression system, DNA encoding a polypeptide is inserted into a pET vector in an orientation designed to allow expression. Since the gene encoding such a polypeptide is under the control of the T7 regulatory signals, expression of the 30 polypeptide is achieved by inducing the expression of T7 RNA polymerase in the host cell. This is typically achieved using host strains that express T7 RNA polymerase in response to IPTG induction. Once produced, recombinant polypeptide is then isolated according to standard methods 35 known in the art, for example, those described herein.

Another bacterial expression system for polypeptide production is the pGEX expression system (Pharmacia). This system employs a GST gene fusion system that is designed proteins with rapid purification and recovery of functional gene products. The protein of interest is fused to the carboxyl terminus of the glutathione S-transferase protein from Schistosoma japonicum and is readily purified from bacterial lysates by affinity chromatography using Glutathione 45 Sepharose 4B. Fusion proteins can be recovered under mild conditions by elution with glutathione. Cleavage of the glutathione S-transferase domain from the fusion protein is facilitated by the presence of recognition sites for site-specific proteases upstream of this domain. For example, pro- 50 teins expressed in pGEX-2T plasmids may be cleaved with thrombin; those expressed in pGEX-3X may be cleaved with

Alternatively, recombinant polypeptides of the invention are expressed in Pichia pastoris, a methylotrophic yeast. 55 Pichia is capable of metabolizing methanol as the sole carbon source. The first step in the metabolism of methanol is the oxidation of methanol to formaldehyde by the enzyme, alcohol oxidase. Expression of this enzyme, which is coded for by the AOX1 gene is induced by methanol. The AOX1 promoter 60 can be used for inducible polypeptide expression or the GAP promoter for constitutive expression of a gene of interest.

Once the recombinant polypeptide of the invention is expressed, it is isolated, for example, using affinity chromatography. In one example, an antibody (e.g., produced as 65 described herein) raised against a polypeptide of the invention may be attached to a column used to isolate the recom30

binant polypeptide. Lysis and fractionation of polypeptideharboring cells prior to affinity chromatography may be performed by standard methods (see, e.g., Ausubel et al., supra). Alternatively, the polypeptide is isolated using a sequence tag, such as a hexahistidine tag (SEQ ID NO: 2314), that binds to nickel column.

Once isolated, the recombinant protein can, if desired, be further purified, e.g., by high performance liquid chromatography (see, e.g., Fisher, Laboratory Techniques In Biochemistry and Molecular Biology, eds., Work and Burdon, Elsevier, 1980). Polypeptides of the invention, particularly short peptide fragments, can also be produced by chemical synthesis (e.g., by the methods described in Solid Phase Peptide Synthesis, 2nd ed., 1984 The Pierce Chemical Co., Rockford, Ill.). These general techniques of polypeptide expression and purification can also be used to produce and isolate useful peptide fragments or analogs (described herein).

Combinatorial Peptide Libraries

In addition to the synthetic solid state production of small peptides, the amino acid sequences of predicted fragments can be expressed and produced recombinantly using a variety of genetically modified organisms following insertion of the relevant DNA into their genome. One such widely used organism is Escherichia coli. Combinatorial biology depends on the ability to link peptides to their encoding DNA and create large libraries of encoded peptides. The methods for generating DNA-encoded peptide libraries can be divided into two groups. In vitro methods use libraries in which the peptides are accessible to exogenous ligands or cells. These libraries can be used in direct in vitro binding selections with cell cultures to enrich for peptides that induce particular phenotypes. In contrast, in vivo methods use peptide libraries that are expressed inside living cells. An interaction between a particular library member and the target protein is detected by virtue of an effect on the host cell, such as a selective growth advantage, or changes to a physical property of the host cell (Pelletier and Sidhu, Curr Opin Biotechnol, 12:340-7, 2001).

To optimize a set of peptides, such as those peptides idenfor high-level expression of genes or gene fragments as fusion 40 tified herein, in vitro methods for creating and testing peptide libraries are suitable. In one embodiment, oligonucleotide directed mutagenesis of initial sequence is used. In another embodiment, a phage is used to display libraries of peptides. Oligonucleotide Directed Mutagenesis

> Oligonucleotide directed mutagenesis can be used in order to modify a single or multiple amino acids that compose the maternal sequence of the predicted anti-angiogenic fragments (Ryu and Nam, Biotechnol Prog, 16:2-16, 2000). Directed mutagenesis is based on the concept that an oligonucleotide encoding a desired mutation is annealed to one strand of a DNA of interest and serves as a primer for initiation of DNA synthesis. In this manner, the mutagenic oligonucleotide is incorporated into the newly synthesized strand. Mutagenic oligonucleotides incorporate at least one base change but can be designed to generate multiple substitutions, insertions or deletions.

> Oligonucleotides can also encode a library of mutations by randomizing the base composition at sites during chemical synthesis resulting in degenerate oligonucleotides. The ability to localize and specify mutations is greatly enhanced by the use of synthetic oligonucleotides hybridized to the DNA insert-containing plasmid vector. The general format for sitedirected mutagenesis includes several steps. Plasmid DNA containing the template of interest (cDNA) is denatured to produce single-stranded regions. A synthetic mutant oligonucleotide is annealed to the target strand. DNA polymerase is used to synthesize a new complementary strand, and finally

DNA ligase is used to seal the resulting nick between the end of the new strand and the oligonucleotide. The resulting heteroduplex is propagated by transformation in *E. coli*. Phage-Displayed Peptide Library Screening

Phage display is one method for in vitro combinatorial 5 biology. The method stems from the observation that peptides fused to certain bacteriophage coat proteins are displayed on the surfaces of phage particles that also contain the cognate DNA (Landon et al., *Curr Drug Discov Technol*, 1:113-32, 2004).

Phage display describes a selection technique in which a library of variants of an initial peptide (e.g., a peptide described herein), is expressed on the outside of a phage virion, while the genetic material encoding each variant resides on the inside. This creates a physical linkage between 15 each variant protein sequence and the DNA encoding it, which allows rapid partitioning based on binding affinity to a given target molecule by an in vitro selection process called panning. In its simplest form, panning is carried out by incubating a library of phage-displayed peptides with a plate 20 containing a culture of cells, such as endothelial cells, washing away the unbound phage, and eluting the specifically bound phage. The eluted phage is then amplified and taken through additional binding/amplification cycles to enrich the pool in favor of specific phenotypes, such as suppression of 25 proliferation, of the cells that are cultured. After 3-4 rounds, individual clones are characterized by DNA sequencing and ELISA.

Libraries of "fusion phages" are rapidly sorted to obtain clones with desired properties and phages can be readily 30 amplified by passage through a bacterial host. Phage display was first demonstrated with the *Escherichia-coli*-specific M13 bacteriophage and this remains the most popular platform. Several other *E. coli* phages have also been adapted for phage display and eukaryotic systems have also been developed.

Screening Assays

Polypeptides and fragments of the invention are useful as targets for the identification of agents that modulate angiogenesis. In particular, the peptides identified herein are typi- 40 cally polypeptide fragments that are hidden within hydrophobic regions of a larger polypeptide. While the entire polypeptide may be pro-angiogenic, the peptides of the invention are typically anti-angiogenic. As such, the activity of these peptides, when exposed to the cellular or extracellular 45 milleau, may reduce the pro-angiogenic function of the larger polypeptide. Where this antagonistic function is undesirable, agents that bind and/or inhibit the biological activity of these peptides are sought. Once identified, such agents are used to enhance angiogenesis. In another approach, anti-angiogenic 50 agents are identified by screening for agents that bind to and enhance the activity of a peptide of the invention. Once identified, such agents are used to reduce angiogenesis.

Alternatively, or in addition, candidate agents may be identified that specifically bind to and inhibit a peptide of the 55 invention. The efficacy of such a candidate compound is dependent upon its ability to interact with the peptide. Such an interaction can be readily assayed using any number of standard binding techniques and functional assays (e.g., those described in Ausubel et al., supra). For example, a candidate 60 compound may be tested in vitro for interaction and binding with a polypeptide of the invention and its ability to modulate angiogenesis may be assayed by any standard assays (e.g., those described herein).

Potential antagonists include organic molecules, peptides, 65 peptide mimetics, polypeptides, nucleic acid ligands, aptamers, and antibodies that bind to a peptide of the invention and

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thereby inhibit or extinguish its activity. Potential antagonists also include small molecules that bind to and occupy the binding site of the polypeptide thereby preventing binding to cellular binding molecules, such that normal biological activity is prevented.

In one particular example, a candidate compound that binds to a pathogenicity polypeptide may be identified using a chromatography-based technique. For example, a recombinant polypeptide of the invention may be purified by standard techniques from cells engineered to express the polypeptide, or may be chemically synthesized, once purified the peptide is immobilized on a column. A solution of candidate compounds is then passed through the column, and a compound specific for the peptide is identified on the basis of its ability to bind to the peptide and be immobilized on the column. To isolate the compound, the column is washed to remove nonspecifically bound molecules, and the compound of interest is then released from the column and collected. Compounds isolated by this method (or any other appropriate method) may, if desired, be further purified (e.g., by high performance liquid chromatography). In addition, these candidate compounds may be tested for their ability to modulate angiogenesis (e.g., as described herein). Compounds isolated by this approach may also be used, for example, as therapeutics to treat or prevent the onset of a disease or disorder characterized by excess or undesirable angiogenesis. Compounds that are identified as binding to peptides with an affinity constant less than or equal to 1 nM, 5 nM, 10 nM, 100 nM, 1 mM or 10 mM are considered particularly useful in the invention.

Methods of the invention are useful for the high-throughput low-cost screening of polypeptides, biologically active fragments or analogs thereof that can be used to modulate angiogenesis. One skilled in the art appreciates that the effects of a candidate peptide on a cell (e.g., an endothelial cell) are typically compared to a corresponding control cell not contacted with the candidate peptide. Thus, the screening methods include comparing the expression profile, phenotype, or biological activity of a cell modulated by a candidate peptide to a reference value of an untreated control cell.

In one example, candidate peptides are added at varying concentrations to the culture medium of an endothelial cell. The survival, tube formation, apoptosis, proliferation, migration of the cell are assayed as indicators of angiogenesis. Peptides that reduce the survival, tube formation, proliferation, or migration of an endothelial cell are identified as useful anti-angiogenic agents. Alternatively, peptides that enhance the survival, tube formation, proliferation, or migration of an endothelial cell are identified as useful angiogenic agents. In another embodiment, the expression of a nucleic acid molecule or polypeptide characteristic of the vasculature is monitored. Typical cell surface markers include the fibronectin extra-domain B, large tenascin-C isoforms, various integrins, VEGF receptors, prostate specific membrane antigen, endoglin and CD44 isoforms and tumor endothelium marker (TEM). Peptides or other agents that alter the expression of such markers are identified as useful modulators of angiogenesis. An agent that reduces the expression of a characteristic polypeptide expressed in the vasculature is considered useful in the invention; such an agent may be used, for example, as a therapeutic to prevent, delay, ameliorate, stabilize, or treat an injury, disease or disorder characterized by an undesirable increase in neovascularization. In other embodiments, agents that increase the expression or activity of a marker characteristically expressed in an endothelial cell are used to prevent, delay, ameliorate, stabilize, or treat an injury, disease or disorder characterized by a reduction in angiogenesis. Agents identified according to the methods described herein maybe

administered to a patient in need of angiogenesis modulation. Where such agents are peptides, such as those described herein, one skilled in the art appreciates that the invention further provides nucleic acid sequences encoding such peptides (e.g., a peptide shown in Tables 1-10).

Test Compounds and Extracts

In general, peptides are identified from large libraries of natural product or synthetic (or semi-synthetic) extracts or chemical libraries or from polypeptide or nucleic acid libraries, according to methods known in the art. Such candidate polypeptides or the nucleic acid molecules encoding them may be modified to enhance biodistribution, protease resistance, or specificity. The modified peptides are then screened for a desired activity (e.g., angiogenesis modulating activity). Those skilled in the field of drug discovery and development 15 will understand that the precise source of test extracts or compounds is not critical to the screening procedure(s) of the invention. Agents used in screens may include known compounds (for example, known polypeptide therapeutics used for other diseases or disorders). Alternatively, virtually any 20 number of unknown chemical extracts or compounds can be screened using the methods described herein. Examples of such extracts or compounds include, but are not limited to, plant-, fungal-, prokaryotic- or animal-based extracts, fermentation broths, and synthetic compounds, as well as the 25 modification of existing polypeptides.

Libraries of natural polypeptides in the form of bacterial, fungal, plant, and animal extracts are commercially available from a number of sources, including Biotics (Sussex, UK), Xenova (Slough, UK), Harbor Branch Oceangraphics Insti- 30 tute (Ft. Pierce, Fla.), and PharmaMar, U.S.A. (Cambridge, Mass.). Such polypeptides can be modified to include a protein transduction domain using methods known in the art and described herein. In addition, natural and synthetically produced libraries are produced, if desired, according to methods 35 known in the art, e.g., by standard extraction and fractionation methods. Examples of methods for the synthesis of molecular libraries can be found in the art, for example in: DeWitt et al., Proc. Natl. Acad. Sci. U.S.A. 90:6909, 1993; Erb et al., Proc. Natl. Acad. Sci. USA 91:11422, 1994; Zuckermann et al., J. 40 Med. Chem. 37:2678, 1994; Cho et al., Science 261:1303, 1993; Carrell et al., Angew. Chem. Int. Ed. Engl. 33:2059, 1994; Carell et al., Angew. Chem. Int. Ed. Engl. 33:2061, 1994; and Gallop et al., J. Med. Chem. 37:1233, 1994. Furthermore, if desired, any library or compound is readily modi-45 fied using standard chemical, physical, or biochemical meth-

Numerous methods are also available for generating random or directed synthesis (e.g., semi-synthesis or total synthesis) of any number of polypeptides, chemical compounds, 50 including, but not limited to, saccharide-, lipid-, peptide-, and nucleic acid-based compounds. Synthetic compound libraries are commercially available from Brandon Associates (Merrimack, N.H.) and Aldrich Chemical (Milwaukee, Wis.). Alternatively, chemical compounds to be used as candidate 55 compounds can be synthesized from readily available starting materials using standard synthetic techniques and methodologies known to those of ordinary skill in the art. Synthetic chemistry transformations and protecting group methodologies (protection and deprotection) useful in synthesizing the 60 compounds identified by the methods described herein are known in the art and include, for example, those such as described in R. Larock, Comprehensive Organic Transformations, VCH Publishers (1989); T. W. Greene and P. G. M. Wuts, Protective Groups in Organic Synthesis, 2nd ed., John 65 Wiley and Sons (1991); L. Fieser and M. Fieser, Fieser and Fieser's Reagents for Organic Synthesis, John Wiley and

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Sons (1994); and L. Paquette, ed., *Encyclopedia of Reagents for Organic Synthesis*, John Wiley and Sons (1995), and subsequent editions thereof.

Libraries of compounds may be presented in solution (e.g., Houghten, *Biotechniques* 13:412-421, 1992), or on beads (Lam, *Nature* 354:82-84, 1991), chips (Fodor, *Nature* 364: 555-556, 1993), bacteria (Ladner, U.S. Pat. No. 5,223,409), spores (Ladner U.S. Pat. No. 5,223,409), plasmids (Cull et al., *Proc Natl Acad Sci USA* 89:1865-1869, 1992) or on phage (Scott and Smith, *Science* 249:386-390, 1990; Devlin, *Science* 249:404-406, 1990; Cwirla et al. *Proc. Natl. Acad. Sci.* 87:6378-6382, 1990; Felici, *J. Mol. Biol.* 222:301-310, 1991; Ladner supra.).

In addition, those skilled in the art of drug discovery and development readily understand that methods for dereplication (e.g., taxonomic dereplication, biological dereplication, and chemical dereplication, or any combination thereof) or the elimination of replicates or repeats of materials already known for their activity should be employed whenever possible.

When a crude extract is found to have angiogenesis modulating activity further fractionation of the positive lead extract is necessary to isolate molecular constituents responsible for the observed effect. Thus, the goal of the extraction, fractionation, and purification process is the careful characterization and identification of a chemical entity within the crude extract that alters angiogenesis (increases or decreases). Methods of fractionation and purification of such heterogeneous extracts are known in the art. If desired, compounds shown to be useful as therapeutics are chemically modified according to methods known in the art.

Therapeutic Methods

Therapeutic polypeptides, peptides, or analogs or fragments thereof, as well as the nucleic acid molecules encoding such molecules are useful for preventing or ameliorating a disease or injury associated with an undesirable increase or decrease in angiogenesis. Diseases and disorders characterized by excess angiogenesis may be treated using the methods and compositions of the invention. Such diseases and disorders include, but are not limited to, neoplasia, hematologic malignancies, rheumatoid arthritis, diabetic retinopathy, agerelated macular degeneration, atherosclerosis, and pathologic obesity. In one embodiment, a peptide of the invention is delivered to one or more endothelial cells at a site of angiogenesis-associated disease or injury.

In other embodiments, a nucleic acid molecule encoding a peptide of the invention is administered to a cell, tissue, or organ in need of a reduction in angiogenesis. If desired, the peptide is expressed as a fusion with a longer polypeptide. The peptide may then be cleaved from the polypeptide to achieve its desired therapeutic effect. Such cleavage is not required where the fusion protein does not interfere with the peptide's biological activity.

Transducing viral (e.g., retroviral, adenoviral, and adenoassociated viral) vectors can be used for somatic cell gene therapy, especially because of their high efficiency of infection and stable integration and expression (see, e.g., Cayouette et al., *Human Gene Therapy* 8:423-430, 1997; Kido et al., *Current Eye Research* 15:833-844, 1996; Bloomer et al., *Journal of Virology* 71:6641-6649, 1997; Naldini et al., *Science* 272:263-267, 1996; and Miyoshi et al., *Proc. Natl. Acad. Sci. U.S.A.* 94:10319, 1997). For example, a full length gene sialidase gene, or a portion thereof, can be cloned into a retroviral vector and expression can be driven from its endogenous promoter, from the retroviral long terminal repeat, or from a promoter specific for a target cell type of interest (e.g. endothelial cell). Other viral vectors that can be used include,

for example, a vaccinia virus, a bovine papilloma virus, or a herpes virus, such as Epstein-Barr Virus (also see, for example, the vectors of Miller, Human Gene Therapy 15-14, 1990; Friedman, *Science* 244:1275-1281, 1989; Eglitis et al., BioTechniques 6:608-614, 1988; Tolstoshev et al., Current 5 Opinion in Biotechnology 1:55-61, 1990; Sharp, The Lancet 337:1277-1278, 1991; Cornetta et al., Nucleic Acid Research and Molecular Biology 36:311-322, 1987; Anderson, Science 226:401-409, 1984; Moen, Blood Cells 17:407-416, 1991; Miller et al., Biotechnology 7:980-990, 1989; Le Gal La Salle 10 et al., Science 259:988-990, 1993; and Johnson, Chest 107:77 S-83S, 1995). Retroviral vectors are particularly well developed and have been used in clinical settings (Rosenberg et al., N. Engl. J. Med 323:370, 1990; Anderson et al., U.S. Pat. No. 5,399,346). Most preferably, a viral vector is used to administer the gene of interest systemically or to a cell at the site of neovascularization.

Non-viral approaches can also be employed for the introduction of therapeutic to a cell of a patient having an angiogenesis related disease. For example, a nucleic acid molecule 20 can be introduced into a cell by administering the nucleic acid in the presence of lipofectin (Feigner et al., *Proc. Natl. Acad. Sci. U.S.A.* 84:7413, 1987; Ono et al., *Neuroscience Letters* 17:259, 1990; Brigham et al., *Am. J. Med. Sci.* 298:278, 1989; Staubinger et al., *Methods in Enzymology* 101:512, 1983), 25 asialoorosomucoid-polylysine conjugation (Wu et al., *Journal of Biological Chemistry* 263:14621, 1988; Wu et al., *Journal of Biological Chemistry* 264:16985, 1989), or by micro-injection under surgical conditions (Wolff et al., *Science* 247:1465, 1990). Preferably the nucleic acids are 30 administered in combination with a liposome and protamine.

Gene transfer can also be achieved using non-viral means involving transfection in vitro. Such methods include the use of calcium phosphate, DEAE dextran, electroporation, and protoplast fusion. Liposomes can also be potentially beneficial for delivery of DNA into a cell. Transplantation of normal genes into the affected tissues of a patient can also be accomplished by transferring a normal nucleic acid into a cultivatable cell type ex vivo (e.g., an autologous or heterologous primary cell or progeny thereof), after which the cell (or its 40 descendants) are injected into a targeted tissue at the site of disease or injury.

cDNA expression for use in such methods can be directed from any suitable promoter (e.g., the human cytomegalovirus (CMV), simian virus 40 (SV40), or metallothionein promoters), and regulated by any appropriate mammalian regulatory element. For example, if desired, enhancers known to preferentially direct gene expression in specific cell types, such as an intestinal epithelial cell, can be used to direct the expression of a nucleic acid. The enhancers used can include, without limitation, those that are characterized as tissue- or cell-specific enhancers. Alternatively, if a genomic clone is used as a therapeutic construct, regulation can be mediated by the cognate regulatory sequences or, if desired, by regulatory sequences derived from a heterologous source, including any of the promoters or regulatory elements described above.

Another therapeutic approach included in the invention involves administration of a recombinant therapeutic, such as a sialidase polypeptide, biologically active fragment, or variant thereof, either directly to the site of a potential or actual 60 disease-affected tissue (for example, by administration to the intestine) or systemically (for example, by any conventional recombinant protein administration technique). The dosage of the administered protein depends on a number of factors, including the size and health of the individual patient. For any particular subject, the specific dosage regimes should be adjusted over time according to the individual need and the

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professional judgment of the person administering or supervising the administration of the compositions. Generally, between 0.1 mg and 100 mg, is administered per day to an adult in any pharmaceutically acceptable formulation.

Pharmaceutical Therapeutics

The invention provides a simple means for identifying compositions (including nucleic acids, peptides, small molecule inhibitors, and mimetics) capable of acting as therapeutics for the treatment of a disease associated with altered levels of angiogenesis. Accordingly, a chemical entity discovered to have medicinal value using the methods described herein is useful as a drug or as information for structural modification of existing compounds, e.g., by rational drug design. Such methods are useful for screening compounds having an effect on a variety of conditions characterized by undesired angiogenesis.

For therapeutic uses, the compositions or agents identified using the methods disclosed herein may be administered systemically, for example, formulated in a pharmaceuticallyacceptable buffer such as physiological saline. Preferable routes of administration include, for example, subcutaneous, intravenous, interperitoneally, intramuscular, or intradermal injections that provide continuous, sustained levels of the drug in the patient. Treatment of human patients or other animals will be carried out using a therapeutically effective amount of a therapeutic agent described herein in a physiologically-acceptable carrier. Suitable carriers and their formulation are described, for example, in Remington's Pharmaceutical Sciences by E. W. Martin. The amount of the therapeutic agent to be administered varies depending upon the manner of administration, the age and body weight of the patient, and with the clinical symptoms of the disease or disorder. Generally, amounts will be in the range of those used for other agents used in the treatment of other diseases associated with alterations in angiogenesis, although in certain instances lower amounts will be needed because of the increased specificity of the compound. A compound is administered at a dosage that controls the clinical or physiological symptoms associated with angiogenesis as determined by a diagnostic method known to one skilled in the art.

It would be advantageous to administer therapeutic peptides in a formulation that would slow their elimination from the circulation through renal filtration, enzymatic degradation, uptake by the reticulo-endothelial system (RES), and accumulation in non-targeted organs and tissues. In addition, methods for administering agents that limits their widespread distribution in non-targeted organs and tissues allows lower concentrations of the agent to be administered reducing adverse side-effects and providing economic benefits. A variety of methods are available to slow the elimination of agents of the invention. In one embodiment, an implantable device is used to provide for the controlled release of an agent described herein. Such devices are known in the art and include, but are not limited to, polymeric gels and microfabricated chips. Some of these devices are already used in the clinic or are being tested in clinical trials (Moses et al., Cancer Cell, 4:337-41, 2003). Various delivery methods for antiangiogenic agents are tissue specific, e.g., intraocular and periocular injection or gene transfer in the eye (Akiyama et al., J Cell Physiol, 2006; Saishin et al., Hum Gene Ther, 16:473-8, 2005). Numerous reviews on the subject of antiangiogenic drug delivery are available.

Enhanced Permeability and Retention Effect

For the treatment of neoplasia or sites of neovascularization, the "enhanced permeability and retention effect" (EPR) constitutes a natural mechanism through which high molecular weight (40 kDa or higher) macromolecules with long

circulation half-lives, including peptides and proteins conjugated with water-soluble polymers, accumulate (Shukla and Krag, Expert Opin Biol Ther, 6:39-54, 2006; Torchilin and Lukyanov, Drug Discov Today, 8:259-66, 2003). This effect occurs because of certain characteristics of those tissues. The first is that tumor or newly formed vasculature, unlike the vasculature of healthy tissues, is permeable to macromolecules with a MW up to 50 kDa or even higher. This allows macromolecules to enter into the interstitial space. Another characteristic is that in the case of many tumors the lymphatic system, which is responsible for the drainage of macromolecules from normal tissues, is impaired. Because of this, macromolecules that have entered a neo-vascularized tissue are retained there for a prolonged time. To enhance the retention of a low MW peptide described herein, the peptide may be conjugated to a suitable polymer or delivered using a micro-reservoir system.

### Peptide and Protein Polymer Conjugation

Mechanisms that increase the MW of a peptide, such as conjugation with polymer chains or concentration of the drug in micro-reservoir systems tend to increase the retention time of the peptide in the tissue (Duncan, *Nat Rev Drug Discov*, 2:347-60, 2003). Moreover, renal filtration and excretion are mainly responsible for the rapid clearance from the systemic circulation of proteins with molecular weights (MW) of 40 kDa or lower. Rapid clearance and increased retention of a peptide of interest can be achieved by conjugating the peptides with water-soluble polymers. Preferably, the peptide-polymer conjugate has a molecular weight of at least about 30 kDA, 35 kDa, 40 kDa, 50 kDa, 75 kDa, or 100 kDa. Additional benefits of peptide and protein-polymer conjugation include increased resistance to enzymatic degradation and reduced immunogenicity.

Even endogenous proteins can be susceptible to protease 35 degradation in the bloodstream and interstitial space or induce an immune response. Enzymatic degradation and an immune response against a protein result in its rapid elimination from the systemic circulation. In addition, the development of an immune response is potentially dangerous because 40 of the possibility of allergic reactions and anaphylactic shock upon repetitive administrations. The mechanism of protein protection by polymer attachment is similar in both cases. Polymer molecules attached to the protein create steric hindrances, which interfere with binding to the active sites of 45 proteases, and antigen-processing cells. Examples of peptide/protein-polymer conjugation include conjugates with poly (ethylene glycol) and conjugates with poly(styrene-co-maleic acid anhydride).

### Conjugates with Poly(Ethylene Glycol)

Several polymers have been used for protein stabilization with varying degrees of success. Poly(ethylene glycol) (PEG) is one widely used polymer for the modification of proteins with therapeutic potential (Thanou and Duncan, *Curr Opin Investig Drugs*, 4:701-9, 2003; Vicent and Duncan, *Trends Biotechnol*, 24:39-47, 2006). This polymer is inexpensive, has low toxicity and has been approved for internal applications by drug regulatory agencies. PEG is commercially available in a variety of molecular weights and in chemically activated, ready-for-use forms for covalent attachment to proteins

Conjugates with Poly(Styrene-Co-Maleic Acid Anhydride)

In some cases, the circulation time of drugs can be increased by conjugating with polymers that are not large enough to prevent renal clearance themselves, but which can 65 attach themselves, with their conjugated drug, to natural long-circulating blood plasma components, such as serum

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albumin or lipoproteins (Thanou and Duncan, *Curr Opin Investig Drugs*, 4:701-9, 2003; Vicent and Duncan, *Trends Biotechnol*, 24:39-47, 2006).

Because of the small size and low molecular weight of the identified anti-angiogenic peptides and the high probability that the conjugated polymers, which are orders of magnitude larger than the peptides, may sterically hinder the activity of the fragments the method of protein conjugation may not be the most efficient method for increasing the retention of the agent in the neo-vascular site. A more attractive scenario is the administration of the peptide in a micro-reservoir delivery system.

Formulation of Pharmaceutical Compositions

The administration of a compound for the treatment of a disease or disorder associated with altered levels of angiogenesis may be by any suitable means that results in a concentration of the therapeutic that, combined with other components, is effective in ameliorating, reducing, or stabilizing a disease or disorder associated with altered levels of angiogenesis (e.g., an amount sufficient to reduce neovascularization). The compound may be contained in any appropriate amount in any suitable carrier substance, and is generally present in an amount of 1-95% by weight of the total weight of the composition. The composition may be provided in a dosage form that is suitable for parenteral (e.g., subcutaneously, intravenously, intramuscularly, or intraperitoneally) administration route. The pharmaceutical compositions may be formulated according to conventional pharmaceutical practice (see, e.g., Remington: The Science and Practice of Pharmacy (20th ed.), ed. A. R. Gennaro, Lippincott Williams & Wilkins, 2000 and Encyclopedia of Pharmaceutical Technology, eds. J. Swarbrick and J. C. Boylan, 1988-1999, Marcel Dekker, New York).

Pharmaceutical compositions according to the invention may be formulated to release the active compound substantially immediately upon administration or at any predetermined time or time period after administration. The latter types of compositions are generally known as controlled release formulations, which include (i) formulations that create a substantially constant concentration of the drug within the body over an extended period of time; (ii) formulations that after a predetermined lag time create a substantially constant concentration of the drug within the body over an extended period of time; (iii) formulations that sustain action during a predetermined time period by maintaining a relatively constant, effective level in the body with concomitant minimization of undesirable side effects associated with fluctuations in the plasma level of the active substance (sawtooth kinetic pattern); (iv) formulations that localize action by, e.g., spatial placement of a controlled release composition adjacent to or in the central nervous system or cerebrospinal fluid; (v) formulations that allow for convenient dosing, such that doses are administered, for example, once every one or two weeks; and (vi) formulations that allow for convenient dosing for metronomic therapy that would require taking small doses of the drug several times a week; (vii) formulations that target a disease or disorder associated with altered levels of angiogenesis by using carriers or chemical derivatives to deliver the therapeutic agent to a particular cell type (e.g., endothelial cell) whose function is perturbed in a disease or disorder associated with altered levels of angiogenesis.

For some applications, controlled release formulations obviate the need for frequent dosing during the day to sustain the plasma level at a therapeutic level.

Any of a number of strategies can be pursued in order to obtain controlled release in which the rate of release outweighs the rate of metabolism of the compound in question.

In one example, controlled release is obtained by appropriate selection of various formulation parameters and ingredients, including, e.g., various types of controlled release compositions and coatings. Thus, the therapeutic is formulated with appropriate excipients into a pharmaceutical composition 5 that, upon administration, releases the therapeutic in a controlled manner. Examples include single or multiple unit tablet or capsule compositions, oil solutions, suspensions, emulsions, microcapsules, microspheres, molecular complexes, nanoparticles, patches, and liposomes.

Micro-Reservoir Delivery Systems

For some applications, micro-reservoir or micro-particulate carriers are used to deliver a peptide of the invention. Such systems include, but are not limited to, liposomes, micelles, polymer micro-particles, and cell ghosts. The use of 15 such carriers results in a much higher ratio of active agent over carrier compared with direct molecular conjugates. They also provide a higher degree of protection against enzymatic degradation and other destructive factors upon parenteral administration because the carrier wall completely isolates 20 drug molecules from the environment. An additional advantage of these carriers is that a single carrier can deliver multiple drug species so that they can be used in combination therapies. All micro-particulates are too large to be lost by renal filtration (Thanou and Duncan, Curr Opin Investig 25 Drugs, 4:701-9, 2003). Exemplary micro-particulate delivery systems include, but are not limited to, liposomes and micelles.

#### Liposomes

Among particulate drug carriers, liposomes are the most 30 extensively studied and possess suitable characteristics for peptide and protein encapsulation. Liposomes are vesicles formed by concentric spherical phospholipid bilayers encapsulating an aqueous space (Moses et al., *Cancer Cell*, 4:337-41, 2003). These particles are biocompatible, biologically 35 inert and cause little toxic or antigenic reactions. Their inner aqueous compartment can be used for encapsulation of peptides and proteins. Many techniques for liposome preparation require only manipulations that are compatible with peptide and protein integrity (Allen and Cullis, *Science*, 303:1818-22, 40 2004). As with other micro-particulate delivery systems, cells of the RES rapidly eliminate conventional liposomes.

In one embodiment, surface-modified long-circulating liposomes grafted with a flexible hydrophilic polymer, such as PEG, are used. This approach prevents plasma protein 45 adsorption to the liposome surface and the subsequent recognition and uptake of liposomes by the RES. Liposomes, in common with protein conjugated macromolecules, can accumulate in tumors of various origins via the EPR effect. Currently, liposomal forms of at least two conventional anticancer drugs, daunorubicin and doxorubicin, are used in the clinic (Torchilin and Lukyanov, *Drug Discov Today*, 8:259-66, 2003).

### Micelles

In another approach, micelles or polymeric micelles, 55 including those prepared from amphiphilic PEG-phospholipid conjugates, may be used to deliver an agent of the invention. Such formulations are of special interest because of their stability (Torchilin and Lukyanov, *Drug Discov Today*, 8:259-66, 2003). These particles are smaller than liposomes and lack the internal aqueous space. To load micelles, peptides can be attached to the surface of these particles or incorporated into them via a chemically attached hydrophobic anchor. An example of a biodegradable micelle developed for delivery of pharmacological agents are the poly{[(cholesteryl oxocarbonylamido ethyl) methyl bis(ethylene) ammonium iodide]ethyl phosphate} (PCEP) micelles (Wen, Mao et

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al., *J Pharm Sci.* 93:2142-57, 2004). Carrying a positive charge in its backbone and a lipophilic cholesterol structure in the side chain, PCEP self-assembles into micelles in aqueous buffer at room temperature with an average size of 60-100 nm. PCEP is an excellent platform for delivering ant-angiogenic agents as by itself shows lower cytotoxicity for endothelial cells than for poly-L-lysine and Lipofectamine. Nanoparticles

An increasing number of agents are delivered with colloidal nanoparticles. Such formulations overcome non-cellular and cellular based mechanisms of resistance and increase the selectivity of agents to target cells while reducing their toxicity in normal tissues. Nanoparticles are typically submicron (<1 µm) colloidal systems. In some embodiments, nanoparticles are made of polymers (biodegradable or not). According to the process used for the preparation of the nanoparticles, nanospheres or nanocapsules can be obtained. Unlike nanospheres (matrix systems in which the drug is dispersed throughout the particles), nanocapsules are vesicular systems in which an agent is confined to an aqueous or oily cavity surrounded by a single polymeric membrane. Nanocapsules are one form of 'reservoir' system.

In some embodiments, nanoparticles are generated using hydrophilic polymers, (poly(ethylene glycol) (PEG), poloxamines, poloxamers, polysaccharides) to efficiently coat a nanoparticle surface. These coatings provide a dynamic 'cloud' of hydrophilic and neutral chains at the particle surface that repels plasma proteins. Hydrophilic polymers are introduced at the surface in two ways, either by adsorption of surfactants or by use of block or branched copolymers.

Parenteral Compositions

The pharmaceutical composition may be administered parenterally by injection, infusion or implantation (subcutaneous, intravenous, intramuscular, intraperitoneal, or the like) in dosage forms, formulations, or via suitable delivery devices or implants containing conventional, non-toxic pharmaceutically acceptable carriers and adjuvants. The formulation and preparation of such compositions are well known to those skilled in the art of pharmaceutical formulation. Formulations can be found in Remington: The Science and Practice of Pharmacy, supra.

Compositions for parenteral use may be provided in unit dosage forms (e.g., in single-dose ampoules), or in vials containing several doses and in which a suitable preservative may be added (see below). The composition may be in the form of a solution, a suspension, an emulsion, an infusion device, or a delivery device for implantation, or it may be presented as a dry powder to be reconstituted with water or another suitable vehicle before use. Apart from the active therapeutic(s), the composition may include suitable parenterally acceptable carriers and/or excipients. The active therapeutic(s) may be incorporated into microspheres, microcapsules, nanoparticles, liposomes, or the like for controlled release. Furthermore, the composition may include suspending, solubilizing, stabilizing, pH-adjusting agents, tonicity adjusting agents, and/or dispersing, agents.

As indicated above, the pharmaceutical compositions according to the invention may be in the form suitable for sterile injection. To prepare such a composition, the suitable active angiogenic modulating therapeutic(s) are dissolved or suspended in a parenterally acceptable liquid vehicle. Among acceptable vehicles and solvents that may be employed are water, water adjusted to a suitable pH by addition of an appropriate amount of hydrochloric acid, sodium hydroxide or a suitable buffer, 1,3-butanediol, Ringer's solution, and isotonic sodium chloride solution and dextrose solution. The aqueous formulation may also contain one or more preserva-

tives (e.g., methyl, ethyl or n-propyl p-hydroxybenzoate). In cases where one of the compounds is only sparingly or slightly soluble in water, a dissolution enhancing or solubilizing agent can be added, or the solvent may include 10-60% w/w of propylene glycol or the like.

Controlled Release Parenteral Compositions

Controlled release parenteral compositions may be in form of aqueous suspensions, microspheres, microcapsules, magnetic microspheres, oil solutions, oil suspensions, or emulsions. Alternatively, the active drug may be incorporated in 10 biocompatible carriers, liposomes, nanoparticles, implants, or infusion devices.

Materials for use in the preparation of microspheres and/or microcapsules are, e.g., biodegradable/bioerodible polymers such as polygalactin, poly-(isobutyl cyanoacrylate), poly(2-15 hydroxyethyl-L-glutam-nine) and, poly(lactic acid). Biocompatible carriers that may be used when formulating a controlled release parenteral formulation are carbohydrates (e.g., dextrans), proteins (e.g., albumin), lipoproteins, or antibodies. Materials for use in implants can be non-biodegradable (e.g., polydimethyl siloxane) or biodegradable (e.g., poly(caprolactone), poly(lactic acid), poly(glycolic acid) or poly(ortho esters) or combinations thereof).

Solid Dosage Forms for Oral Use

Formulations for oral use include tablets containing the 25 active ingredient(s) in a mixture with non-toxic pharmaceutically acceptable excipients. Such formulations are known to the skilled artisan. Excipients may be, for example, inert diluents or fillers (e.g., sucrose, sorbitol, sugar, mannitol, microcrystalline cellulose, starches including potato starch, 30 calcium carbonate, sodium chloride, lactose, calcium phosphate, calcium sulfate, or sodium phosphate); granulating and disintegrating agents (e.g., cellulose derivatives including microcrystalline cellulose, starches including potato starch, croscarmellose sodium, alginates, or alginic acid); binding 35 agents (e.g., sucrose, glucose, sorbitol, acacia, alginic acid, sodium alginate, gelatin, starch, pregelatinized starch, microcrystalline cellulose, magnesium aluminum silicate, carboxymethylcellulose sodium, methylcellulose, hydroxypropyl methylcellulose, ethylcellulose, polyvinylpyrrolidone, or 40 polyethylene glycol); and lubricating agents, glidants, and antiadhesives (e.g., magnesium stearate, zinc stearate, stearic acid, silicas, hydrogenated vegetable oils, or talc). Other pharmaceutically acceptable excipients can be colorants, flavoring agents, plasticizers, humectants, buffering agents, and 45 the like.

The tablets may be uncoated or they may be coated by known techniques, optionally to delay disintegration and absorption in the gastrointestinal tract and thereby providing a sustained action over a longer period. The coating may be 50 adapted to release the active drug in a predetermined pattern (e.g., in order to achieve a controlled release formulation) or it may be adapted not to release the active drug until after passage of the stomach (enteric coating). The coating may be a sugar coating, a film coating (e.g., based on hydroxypropyl 55 methylcellulose, methylcellulose, methyl hydroxyethylcellulose, hydroxypropylcellulose, carboxymethylcellulose, acrylate copolymers, polyethylene glycols and/or polyvinylpyrrolidone), or an enteric coating (e.g., based on methacrylic acid copolymer, cellulose acetate phthalate, hydroxypropyl 60 methylcellulose phthalate, hydroxypropyl methylcellulose acetate succinate, polyvinyl acetate phthalate, shellac, and/or ethylcellulose). Furthermore, a time delay material such as, e.g., glyceryl monostearate or glyceryl distearate may be employed.

The solid tablet compositions may include a coating adapted to protect the composition from unwanted chemical 42

changes, (e.g., chemical degradation prior to the release of the active angiogenic modulating therapeutic). The coating may be applied on the solid dosage form in a similar manner as that described in *Encyclopedia of Pharmaceutical Technology*, supra.

At least two active angiogenic modulating therapeutics may be mixed together in the tablet, or may be partitioned. In one example, the first active in angiogenic modulating therapeutic is contained on the inside of the tablet, and the second active angiogenic modulating therapeutic is on the outside, such that a substantial portion of the second angiogenic modulating therapeutic is released prior to the release of the first angiogenic modulating therapeutic.

Formulations for oral use may also be presented as chewable tablets, or as hard gelatin capsules wherein the active ingredient is mixed with an inert solid diluent (e.g., potato starch, lactose, microcrystalline cellulose, calcium carbonate, calcium phosphate or kaolin), or as soft gelatin capsules wherein the active ingredient is mixed with water or an oil medium, for example, peanut oil, liquid paraffin, or olive oil. Powders and granulates may be prepared using the ingredients mentioned above under tablets and capsules in a conventional manner using, e.g., a mixer, a fluid bed apparatus or a spray drying equipment.

Controlled Release Oral Dosage Forms

Controlled release compositions for oral use are constructed to release the active angiogenic modulating therapeutic by controlling the dissolution and/or the diffusion of the active substance. Dissolution or diffusion controlled release can be achieved by appropriate coating of a tablet, capsule, pellet, or granulate formulation of compounds, or by incorporating the compound into an appropriate matrix. A controlled release coating may include one or more of the coating substances mentioned above and/or, e.g., shellac, beeswax, glycowax, castor wax, carnauba wax, stearyl alcohol, glyceryl monostearate, glyceryl distearate, glycerol palmitostearate, ethylcellulose, acrylic resins, dl-polylactic acid, cellulose acetate butyrate, polyvinyl chloride, polyvinyl acetate, vinyl pyrrolidone, polyethylene, polymethacrylate, methylmethacrylate, 2-hydroxymethacrylate, methacrylate hydrogels, 1,3 butylene glycol, ethylene glycol methacrylate, and/or polyethylene glycols. In a controlled release matrix formulation, the matrix material may also include, e.g., hydrated methylcellulose, carnauba wax and stearyl alcohol, carbopol 934, silicone, glyceryl tristearate, methyl acrylatemethyl methacrylate, polyvinyl chloride, polyethylene, and/ or halogenated fluorocarbon.

A controlled release composition containing one or more therapeutic compounds may also be in the form of a buoyant tablet or capsule (i.e., a tablet or capsule that, upon oral administration, floats on top of the gastric content for a certain period of time). A buoyant tablet formulation of the compound(s) can be prepared by granulating a mixture of the compound(s) with excipients and 20-75% w/w of hydrocolloids, such as hydroxyethylcellulose, hydroxypropylcellulose, or hydroxypropylmethylcellulose. The obtained granules can then be compressed into tablets. On contact with the gastric juice, the tablet forms a substantially water-impermeable gel barrier around its surface. This gel barrier takes part in maintaining a density of less than one, thereby allowing the tablet to remain buoyant in the gastric juice.

Polymeric Controlled-Release Implants

In another embodiment, an agent of the invention is delivered by implanting a biodegradable polymeric controlledrelease device that stores the pharmaceutical agent and allows its delivery via diffusion into the surrounding tissue. Controlled release devices include Norplant and Gliadel, which

are used clinically for the prevention of pregnancy and for brain tumor therapy, respectively. Local delivery of pro- or anti-angiogenic factors can be accomplished by encapsulating the agent within a biocompatible polymer matrix. The controlled-release polymer system is then implanted at the 5 desired tissue site, where it releases the soluble factor directly into the interstitial space of the tissue. The diffusible agent can influence the survival or function of damaged cells within the local tissue, or provide a signal that elicits cell proliferation and migration or apoptosis and suppression of migration 10 within the tissue region.

Controlled release implants are typically composed of inert, biocompatible polymers, such as poly(ethylene-co-vinyl acetate) (EVAc), or biodegradable polymers, such as poly (lactide-co-glycolide) (PLGA) (Torchilin and Lukyanov, 15 Drug Discov Today, 8:259-66, 2003). EVAc-matrix systems have been used to release protein hormones, growth factors, antibodies, antigens and DNA. EVAc matrices allow a high degree of control over agent release, versatility in allowing the release of a wide range of agents, and good retention of 20 biological activity. Biodegradable polymers have also been used to release growth factors, protein hormones, antibodies, antigens and DNA. Biodegradable materials disappear from the implant site after protein release. Polymer gels might also be useful for topical or localized protein delivery. Systems 25 that release multiple protein factors are also possible (Saltzman and Olbricht, Nat Rev Drug Discov, 1:177-86, 2002; Torchilin and Lukyanov, Drug Discov Today, 8:259-66, 2003).

Biodegradable polymers include non-water-soluble poly- 30 mers that are degraded by surface or bulk erosion in addition to water-soluble gels that dissolve and are cleared from the body without undergoing a decrease in molecular weight. There are many different types of biodegradable polymers that can potentially be used in the preparation of peptide 35 delivery systems. They include both naturally derived and synthetic materials.

### Biocompatibility of Polymeric Systems

Polymers used as drug delivery systems for protein pharmaceuticals need to exhibit biocompatible characteristics in 40 terms of both the polymer's effect on the organism receiving the drug delivery system and the polymer's effect on the protein to be delivered. Several aspects of a polymeric delivery system ultimately contribute to its overall biocompatibility, or lack thereof. The polymer itself, which consists of a 45 repeating monomeric species, may potentially be antigenic, carcinogenic, or toxic or have some inherent incompatibility with organisms. The shape of an implanted material has been implicated in its biocompatibility as well, smooth surfaces being less irritating and more biocompatible than rough sur- 50 faces (Saltzman and Olbricht, Nat Rev Drug Discov, 1:177-86, 2002).

### Pharmaceutical Stability

Interactions between proteins and polymeric materials appear to be protein and polymer specific. At issue are the 55 protein molecular weight, which is an important parameter with regard to diffusion characteristics and the iso-electric point of the protein (and polymer as well in some cases), which governs charge-charge interactions (protein-polymer and protein-protein). Moreover the presence of cysteines on 60 the protein may facilitate the formation of intermolecular (i.e., protein-polymer) disulfide bonds. Furthermore, the primary amino acid sequence of the protein may be rendered susceptible to chemical modification in association with a polymeric material. The presence or absence of carbohydrates on the protein may enhance or prevent interaction with polymeric materials and affect the protein's hydrodynamic

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volume. The relative hydrophobicity of a protein could interact with hydrophobic sites on a polymer. Finally the heterogeneity of protein pharmaceuticals often exists for proteins produced by recombinant methods (Bilati et al., Eur J Pharm Biopharm, 59:375-88, 2005; Gombotz and Pettit, Bioconjug Chem, 6:332-51, 1995; Saltzman and Olbricht, Nat Rev Drug Discov. 1:177-86, 2002).

**Bulk Erosion Polymers** 

Poly(Lactic-Co-Glycolic Acid)

Poly(lactic-co-glycolic acid) (PLGA) has been used successfully for several decades in biodegradable structures and more recently as drug delivery micro-carriers, and as a result of the extended use, much is known about their biocompatibility and physicochemical characteristics. PLGA copolymers are well suited for use in delivery systems since they can be fabricated into a variety of morphologies including films, rods, spheres by solvent casting, compression molding and solvent evaporation techniques. Examples of peptide drug delivery systems made from PLGA copolymers, have successfully met FDA approval and they are available as marketed products are Lupron Depot, Zoladex and Decapeptyl (Frokjaer and Otzen, Nat Rev Drug Discov, 4:298-306, 2005). Block Copolymers of PEG and PLA

Copolymers of PEG and PLA have been synthesized for use in delivery systems. The net result is a biodegradable polymer with a reduced amount of hydrophobicity that is an inherent property of PLA systems. These copolymer systems can be composed of random blocks of the two polymers, two blocks in which case the molecules are amphiphilic, or triblocks in which hydrophilic microphases are present. Peptides that are incorporated into devices made from these copolymers are less likely to adsorb to the delivery system through hydrophobic interactions. The polymers were shown to swell very rapidly due to microphase separation, and degradation occurred over 2-3 weeks (Bilati et al., Eur J Pharm Biopharm, 59:375-88, 2005; Gombotz and Pettit, Bioconjug Chem, 6:332-51, 1995).

Poly(Cyanoacrylates)

Poly(cyanoacrylates) have received attention as delivery systems for proteins and peptides. They undergo spontaneous polymerization at room temperature in the presence of water, and their erosion has been shown to be controlled by the length of the monomer chain and the pH. Once formed, the polymer is slowly hydrolyzed, leading to a chain scission and liberation of formaldehyde. While the polymers are not toxic. the formaldehyde released as the degradation byproduct does create a toxicity concern. A characteristic example of their use are delivery systems for insulin prepared by the interfacial emulsion polymerization of alkyl cyanoacrylate (Gombotz and Pettit, Bioconjug Chem, 6:332-51, 1995).

Surface Erosion Polymers

Poly(Anhydrides)

Poly(anhydrides) represent a class of surface eroding polymers. Hydrolysis of the anhydride bond is suppressed by acid, which results in an inhibition of bulk erosion by the acidity of the carboxylic acid products of the polymer hydrolysis process. By varying the ratio of their hydrophobic components, one can control degradation rates ranging from days to years. Several proteins have been successfully incorporated into, and released, from poly-(anhydride) delivery systems. The incorporation of insulin and myoglobin has successfully been achieved in poly(anhydride) microspheres using both a hotmelt microencapsulation technique or microencapsulation by solvent removal (Gombotz and Pettit, Bioconjug Chem, 6:332-51, 1995).

Poly(Ortho Esters)

Poly(ortho esters) are another example of surface-eroding polymers that have been developed for drug delivery systems. Several proteins and peptides have been incorporated into poly(ortho-ester) delivery systems including the LHRH analog nafarelin, insulin and lysozyme.

The use of biodegradable hydrogels as delivery systems for proteins is of particular interest due to their biocompatibility and their relative inertness toward protein drugs (Gombotz and Pettit, Bioconjug Chem, 6:332-51, 1995). Hydrogels are the only class of polymer that can enable a protein to permeate through the continuum of the carrier. The initial release rate of proteins from biodegradable hydrogels is therefore generally diffusion controlled through the aqueous channels of the gel and is inversely proportional to the molecular weight of the protein. Once polymer degradation occurs, and if protein still remains in the hydrogel, erosion-controlled release may contribute to the system. Several disadvantages must be consid- 20 ered when using a biodegradable hydrogel system for the release of proteins. Their ability to rapidly swell with water can lead to very fast release rates and polymer degradation rates. In addition, hydrogels can rapidly decrease in mechanical strength upon swelling with water. Examples of hydrogels 25 include, pluronic polyols, poly(vinyl alcohol), poly(vinylpyrrolidone), malein anhydride, callulose, hyaluronic acid derivatives, alginate, collagens, gelatin, starches and dextrans.

Selective Drug Delivery

Selective delivery of therapeutic agents includes any methodology by which the functional concentration of drug is higher at the target site than in normal tissue. A wide variety of methods may fall under the category of "selective delivery," including interventions as simple and mechanical as 35 selective vascular administration in which the drug is physically isolated in a neovascularized area. An example of that type of mechanical selectivity is also the EPR effect.

Most strategies, however, are pharmaceutical. In these approaches, the differences in the biochemical and physi-ological nature of normal and the targeted cells and their microenvironment are exploited for selective delivery. In one embodiment, a carrier is used to deliver a peptide of the invention that because of its physical properties, accumulates preferentially at a target site. In another embodiment, a ligand 45 is conjugated to a peptide of the invention that binds to a tissue-associated antigen. In another embodiment, an agent of the invention is maintained in an inactive form that can be activated preferentially at the tissue site. In yet another embodiment, external energy irradiation is used to release a 50 peptide at the delivery site.

A variety of technologies using combinations of different approaches are constantly being developed for selective delivery of therapeutics. These delivery systems employ different targets such as cancer cell and neovascular antigens, 55 hypoxia, or high osmotic pressure; targeting agents such as monoclonal antibodies (mAbs), single chain variable fragments (scFvs), peptides and oligonucleotides; effectors like chemical or biological toxins, radioisotopes, genes, enzymes, immunomodulators, oligonucleotides, imaging and diagnos- 60 tic agents; vehicles the already mentioned colloidal systems, including liposomes, emulsions, micelles, nanoparticles, polymer conjugates or implants; and drug-releasing switches such as systems that utilize thermal, radiation, ultrasound or magnetic fields (Allen and Cullis, Science, 303:1818-22, 65 2004; Gombotz and Pettit, Bioconjug Chem, 6:332-51, 1995; Moses et al., Cancer Cell, 4:337-41, 2003; Neri and Bicknell,

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Nat Rev Cancer, 5:436-46, 2005; Saltzman and Olbricht, Nat Rev Drug Discov, 1:177-86, 2002).

**Tumor Marker Targeting** 

The advent of aptamer and antibody technology has facilitated the use of cancer-specific monoclonal antibodies and aptamers to deliver peptides of the invention to a selected target tissue. Of special interest are antibodies and aptamers that target, in vivo, tumor endothelium. Those targets include, but are not limited to, the fibronectin extra-domain B, large tenascin-C isoforms, various integrins, VEGF receptors, prostate specific membrane antigen, endoglin and CD44 isoforms (Shukla and Krag, Expert Opin Biol Ther, 6:39-54, 2006). Alternatively, the tumor itself may be targeted, exemplary tumor markers include CA-125, gangliosides G(D2), G(M2) and G(D3), CD20, CD52, CD33, Ep-CAM, CEA, bombesin-like peptides, PSA, HER2/neu, epidermal growth factor receptor, erbB2, erbB3, erbB4, CD44v6, Ki-67, cancer-associated mucin, VEGF, VEGFRs (e.g., VEGFR3), estrogen receptors, Lewis-Y antigen, TGFβ1, IGF-1 receptor, EGFα, c-Kit receptor, transferrin receptor, IL-2R and CO17-1A. Aptamers and antibodies of the invention can recognize tumors derived from a wide variety of tissue types, including, but not limited to, breast, prostate, colon, lung, pharynx, thyroid, lymphoid, lymphatic, larynx, esophagus, oral mucosa, bladder, stomach, intestine, liver, pancreas, ovary, uterus, cervix, testes, dermis, bone, blood and brain. In the context of the present invention, a tumor cell is a neoplastic (e.g., cancer) cell or a mass of cancer cells, which can also encompass cells that support the growth and/or propagation of a cancer cell, such as vasculature and/or stroma, but not necessarily macrophages. For instance, therefore, the present invention envisages compositions and methods for reducing growth of a tumor cell in a subject, wherein antibodies or aptamers bind with specificity to cell surface epitopes (or epitopes of receptor-binding molecules) of a cancer cell or a cell that is involved in the growth and/or propagation of a cancer cell such as a cell comprising the vasculature of a tumor or blood vessels that supply tumors and/or stromal cells. Methods of this invention are particularly suitable for administration to humans with neoplastic diseases. Antibodies

Antibodies are well known to those of ordinary skill in the science of immunology. Particularly useful in the methods of the invention are antibodies that specifically bind a polypeptide that is expressed in a tumor or endothelial cell. As used herein, the term "antibody" means not only intact antibody molecules, but also fragments of antibody molecules that retain immunogen binding ability. Such fragments are also well known in the art and are regularly employed both in vitro and in vivo. Accordingly, as used herein, the term "antibody" means not only intact immunoglobulin molecules but also the well-known active fragments F(ab')<sub>2</sub>, and Fab. F(ab')<sub>2</sub>, and Fab fragments which lack the Fc fragment of intact antibody, clear more rapidly from the circulation, and may have less non-specific tissue binding of an intact antibody (Wahl et al., J. Nucl. Med. 24:316-325, 1983). The antibodies of the invention comprise whole native antibodies, bispecific antibodies; chimeric antibodies; Fab, Fab', single chain V region fragments (scFv) and fusion polypeptides.

In one embodiment, an antibody that binds polypeptide is monoclonal. Alternatively, the antibody is a polyclonal antibody. The preparation and use of polyclonal antibodies are also known to the skilled artisan. The invention also encompasses hybrid antibodies, in which one pair of heavy and light chains is obtained from a first antibody, while the other pair of heavy and light chains is obtained from a different second

antibody. Such hybrids may also be formed using humanized heavy and light chains. Such antibodies are often referred to as "chimeric" antibodies.

In general, intact antibodies are said to contain "Fc" and "Fab" regions. The Fc regions are involved in complement 5 activation and are not involved in antigen binding. An antibody from which the Fc' region has been enzymatically cleaved, or which has been produced without the Fc' region, designated an "F(ab')2" fragment, retains both of the antigen binding sites of the intact antibody. Similarly, an antibody from which the Fc region has been enzymatically cleaved, or which has been produced without the Fc region, designated an "Fab" fragment, retains one of the antigen binding sites of the intact antibody. Fab' fragments consist of a covalently bound antibody light chain and a portion of the antibody 15 heavy chain, denoted "Fd." The Fd fragments are the major determinants of antibody specificity (a single Fd fragment may be associated with up to ten different light chains without altering antibody specificity). Isolated Fd fragments retain the ability to specifically bind to immunogenic epitopes.

Antibodies can be made by any of the methods known in the art utilizing a peptide of the invention (e.g., a peptide shown in Tables 1-10), or immunogenic fragments thereof, as an immunogen. One method of obtaining antibodies is to immunize suitable host animals with an immunogen and to 25 follow standard procedures for polyclonal or monoclonal antibody production. The immunogen will facilitate presentation of the immunogen on the cell surface. Immunization of a suitable host can be carried out in a number of ways. Nucleic acid sequences encoding a polypeptide described herein, or 30 immunogenic fragments thereof, can be provided to the host in a delivery vehicle that is taken up by immune cells of the host. The cells will in turn express the receptor on the cell surface generating an immunogenic response in the host. Alternatively, nucleic acid sequences encoding a peptide of 35 the invention (e.g., a peptide shown in Tables 1-10), or immunogenic fragments thereof, can be expressed in cells in vitro, followed by isolation of the polypeptide and administration of the receptor to a suitable host in which antibodies are raised.

Using either approach, antibodies can then be purified from 40 the host. Antibody purification methods may include salt precipitation (for example, with ammonium sulfate), ion exchange chromatography (for example, on a cationic or anionic exchange column preferably run at neutral pH and eluted with step gradients of increasing ionic strength), gel 45 filtration chromatography (including gel filtration HPLC), and chromatography on affinity resins such as protein A, protein G, hydroxyapatite, and anti-immunoglobulin.

Antibodies can be conveniently produced from hybridoma cells engineered to express the antibody. Methods of making 50 hybridomas are well known in the art. The hybridoma cells can be cultured in a suitable medium, and spent medium can be used as an antibody source. Polynucleotides encoding the antibody of interest can in turn be obtained from the hybridoma that produces the antibody, and then the antibody may 55 be produced synthetically or recombinantly from these DNA sequences. For the production of large amounts of antibody, it is generally more convenient to obtain an ascites fluid. The method of raising ascites generally comprises injecting hybridoma cells into an immunologically naive histocompatible or 60 immunotolerant mammal, especially a mouse. The mammal may be primed for ascites production by prior administration of a suitable composition; e.g., Pristane.

Monoclonal antibodies (Mabs) produced by methods of the invention can be "humanized" by methods known in the 65 art. "Humanized" antibodies are antibodies in which at least part of the sequence has been altered from its initial form to 48

render it more like human immunoglobulins. Techniques to humanize antibodies are particularly useful when non-human animal (e.g., murine) antibodies are generated. Examples of methods for humanizing a murine antibody are provided in U.S. Pat. Nos. 4,816,567, 5,530,101, 5,225,539, 5,585,089, 5,693,762 and 5,859,205.

Aptamers

Nucleic acid aptamers are single-stranded nucleic acid (DNA or RNA) ligands that function by folding into a specific globular structure that dictates binding to target proteins or other molecules with high affinity and specificity, as described by Osborne et al., Curr. Opin. Chem. Biol. 1:5-9, 1997; and Cerchia et al., FEBS Letters 528:12-16, 2002. By "aptamer" is meant a single-stranded polynucleotide that binds to a protein. Desirably, the aptamers are small, approximately ~15 KD. The aptamers are isolated from libraries consisting of some  $10^{14}$ - $10^{15}$  random oligonucleotide sequences by a procedure termed SELEX (systematic evolution of ligands by exponential enrichment). See Tuerk et al., 20 Science, 249:505-510, 1990; Green et al., Methods Enzymology. 75-86, 1991; Gold et al., Annu. Rev. Biochem., 64: 763-797, 1995; Uphoff et al., Curr. Opin. Struct. Biol., 6: 281-288, 1996. Methods of generating aptamers are known in the art and are described, for example, in U.S. Pat. Nos. 6,344,318, 6,331,398, 6,110,900, 5,817,785, 5,756,291, 5,696,249, 5,670,637, 5,637,461, 5,595,877, 5,527,894, 5,496,938, 5,475,096, 5,270,163, and in U.S. Patent Application Publication Nos. 20040241731, 20030198989, 20030157487, and 20020172962.

An aptamer of the invention is capable of binding with specificity to a polypeptide expressed by a cell of interest (e.g., a tumor cell or an endothelial cell supplying a tumor). "Binding with specificity" means that non-tumor polypeptides are either not specifically bound by the aptamer or are only poorly bound by the aptamer. In general, aptamers typically have binding constants in the picomolar range. Particularly useful in the methods of the invention are aptamers having apparent dissociation constants of 1, 10, 15, 25, 50, 75, or 100 nM.

In one embodiment, an antigen expressed on a blood vessel supplying a tumor is the molecular target of the aptamer. Because aptamers can act as direct antagonists of the biological function of proteins, aptamers that target such polypeptide can be used to modulate angiogenesis, vasculogenesis, blood vessel stabilization or remodeling. The therapeutic benefit of such aptamers derives primarily from the biological antagonism caused by aptamer binding.

The invention encompasses stabilized aptamers having modifications that protect against 3' and 5' exonucleases as well as endonucleases. Such modifications desirably maintain target affinity while increasing aptamer stability in vivo. In various embodiments, aptamers of the invention include chemical substitutions at the ribose and/or phosphate and/or base positions of a given nucleobase sequence. For example, aptamers of the invention include chemical modifications at the 2' position of the ribose moiety, circularization of the aptamer, 3' capping and 'spiegelmer' technology. Aptamers having A and G nucleotides sequentially replaced with their 2'-OCH3 modified counterparts are particularly useful in the methods of the invention. Such modifications are typically well tolerated in terms of retaining aptamer affinity and specificity. In various embodiments, aptamers include at least 10%, 25%, 50%, or 75% modified nucleotides. In other embodiments, as many as 80-90% of the aptamer's nucleotides contain stabilizing substitutions. In other embodiments, 2'-OMe aptamers are synthesized. Such aptamers are desirable because they are inexpensive to synthesize and

natural polymerases do not accept 2'-OMe nucleotide triphosphates as substrates so that 2'-OMe nucleotides cannot be recycled into host DNA. A fully 2'-O-methyl aptamer, named ARC245, was reported to be so stable that degradation could not be detected after 96 hours in plasma at 37° C. or after 5 autoclaving at 125° C. Using methods described herein, aptamers will be selected for reduced size and increased stability. In one embodiment, aptamers having 2'-F and 2'-OCH<sub>3</sub> modifications are used to generate nuclease resistant aptamers. Other modifications that stabilize aptamers are 10 known in the art and are described, for example, in U.S. Pat. No. 5,580,737; and in U.S. Patent Application Publication Nos. 20050037394, 20040253679, 20040197804, and 20040180360.

Using standard methods tumor markers or endothelial call- 15 specific aptamers can be selected that bind virtually any tumor marker or endothelial cell-expressed polypeptide known in the art.

The Fibronectin Extra-Domain B (EDB)

Fibronectin is a large glycoprotein that is present in large 20 amounts in the plasma and tissues. EDB is a 91-amino-acid type III homology domain that becomes inserted into the fibronectin molecule under tissue-remodeling conditions by a mechanism of alternative splicing at the level of the primary transcript. EDB is essentially undetectable in healthy adult 25 individuals. EDB-containing fibronectin is abundant in many aggressive solid tumors and in neo-vascularized endothelium, and displays either predominantly vascular or diffuse stromal patterns of expression, depending on the tissue.

Large Tenascin-C Isoforms

Tenascins are a family of four extracellular matrix glycoproteins that are found in vertebrates. They are typically present in many different connective tissues. Tenascins contribute to matrix structure and influence the behavior of cells that are in contact with the extracellular matrix. Several iso- 35 forms of tenascin-C can be generated as a result of different patterns of alternative splicing in the region between domains A1 and D. It has been known for some time that spliced isoforms containing extra domains are tumor-associated antigens, which show a more restricted pattern of expression in 40 normal tissues compared with the "small" tenascin isoforms. The C domain of tenascin-C is the extra domain that shows the most restricted pattern of expression. In normal adult tissue it is undetectable by immunohistochemistry and northern-blot analysis, but it is strongly expressed in aggressive 45 brain tumors and some lung tumors, with a prominent perivascular pattern of staining.

Integrins

During vascular remodeling and angiogenesis, endothelial cells show increased expression of several cell-surface molecules that potentiate cell invasion and proliferation. One such molecule is the integrin  $\alpha v$ - $\beta 3$ , which has a key role in endothelial cell survival during angiogenesis in vivo and which might serve as a target for therapeutic molecules, particularly those that require internalization in endothelial cells. 55 Monoclonal antibodies to the  $\alpha v$ - $\beta 3$  have been shown to display anti-angiogenic activities and to preferentially stain tumor blood vessels.

VEGFs and their Receptors

VEGFs represent a class of proteins that promote angiogenesis, increase vascular permeability and contribute to endothelial-cell survival in blood and lymphatic vessels. The contribution of VEGFA to cancer progression has been highlighted by the recent approval of the humanized anti-VEGF monoclonal antibody bevacizumab (Avastin; Genentech) for 65 first-line cancer treatment. The overexpression of VEGFs and VEGF receptors in tumors is well documented. The selective

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tumor localization of monoclonal antibodies to VEGFA, VEGF receptor 2 and the VEGFA-VEGF receptor 2 complex can be used as an excellent selectivity mechanism for targeting the angiogenic vasculature.

Prostate-Specific Membrane Antigen

Prostate-specific membrane antigen (PSMA) is a membrane glycoprotein with proteolytic activity. PSMA is predominantly expressed in the prostate and serum concentrations are often increased in patients with prostate cancer. Several studies have reported overexpression of PSMA in the neo-vasculature of different solid tumors, whereas expression in normal vasculature is limited to some vessels of the breast, duodenum, kidney and prostate. Endoglin

Endoglin (CD105) is a transforming growth factor-beta (TGF) co-receptor that is overexpressed in tumor neo-vasculature and is used as a marker for the tumor endothelium. CD44 Isoforms

CD44 is a cell-surface receptor of great molecular heterogeneity, which is due to both alternative splicing and extensive post-translational modification. The radio-labeled monoclonal antibody TES-23, which is specific to an isoform of CD44, has shown impressive performance in tumor-targeting experiments in animal models. TES-23 recognizes a widely distributed form of CD44 that lacks variant exons, known as CD44H.

Tumor Endothelial Markers (TEMs)

TEMs is a family of genes encoding proteins that serve as tumor endothelial markers (Carson-Walter, Watkins, et al, *Cancer Res.* 61:6649-55, 2001). These genes display elevated expression during tumor angiogenesis. From both biological and clinical points of view, TEMs associated with the cell surface membrane are of particular interest. Accordingly, four such genes are characterized, TEM1, TEM5, TEM7, and TEM8, all of which contain putative transmembrane domains. TEM5 appears to be a seven-pass transmembrane receptor, whereas TEM1, TEM7, and TEM8 span the membrane once. Three of these TEMs (TEM1, TEM5, and TEM8) are abundantly expressed in tumor vessels in mouse tumors, embryos, and adult tissues as well as in the vasculature of the developing mouse embryo. The expression of these TEMs in normal adult mice tissues is undetectable.

Selective Delivery Through Pro-Drug Activation

Selective delivery of agents of the invention can be achieved by administering a pro-drug form that is converted into an active drug at the target site. Most pro-drugs are designed to have a "trigger," "linker" and "effector." The "trigger," following the tissue-specific metabolism, modifies the "linker," resulting in an activation of the "effector." There are several mechanisms potentially exploitable for selective activation. Some utilize unique aspects of the tissue physiology, such as selective enzyme expression or hypoxia in the case of tumors, whereas others are based on tissue antigenspecific delivery techniques.

Targeting Secreted Enzymes from Cells

The approach uses pro-drugs that are "hidden" from the cells until cleaved by an enzyme produced and secreted preferentially by the cells. A typical example of an enzyme used for pro-drug activation is MMP-9.

Targeting Tumor Hypoxia

Advances in the chemistry of bio-reductive drug activation have led to the design of hypoxia-selective drug delivery systems. These pro-drugs initially undergo one-electron reduction by reductases to give the radical anion, which in normal cells are re-oxidized to the parent compound, but in hypoxic tumor cells they are further reduced to more hydrophilic species and trapped inside. These drugs can be selec-

tively delivered to tumors with defined hypoxic fractions rich in the required activating enzymes.

Antibody-Directed Enzyme Pro-Drug Therapy

Antibody-directed pro-drug therapy (ADEPT) is a 2-step approach in which first the antibody-enzyme construct is administered intravenously. This is composed of an antibody against a tissue-specific target linked to an enzyme that activates a pro-drug. In the second step, after the unbound antibody-enzyme conjugate construct is cleared from the circulation, a pro-drug is administered intravenously. The pro-drug is an agent that has been rendered less active by chemical addition of enzyme-cleavable moieties. The pro-drug is converted to an active form by the tumor-bound antibody-enzyme, which results in local accumulation of the fully active form of the agent.

External Energy-Controlled Delivery

Some selective delivery strategies involve focusing external energy for concentrating or delivering therapeutics at the tissue site. A variety of delivery systems in this category are in the experimental stage, although some have been used in 20 clinical trials as well. Those strategies include selective delivery through photodynamic therapy, magnetically targeted delivery, selective delivery through X-ray exposure, radiation-induced selective delivery and ultrasound-guided delivery.

### Methods of Ocular Delivery

The compositions of the invention (e.g., a peptide of the invention shown in Tables 1-10) are also particularly suitable for treating ocular diseases, such as age-related macular degeneration, choroidal neovascularization, persistent hyperplastic vitreous syndrome, diabetic retinopathy, and retinopathy of prematurity that are characterized by excess angiogenesis.

In one approach, the compositions of the invention are administered through an ocular device suitable for direct 35 implantation into the vitreous of the eye. The compositions of the invention may be provided in sustained release compositions, such as those described in, for example, U.S. Pat. Nos. 5,672,659 and 5,595,760. Such devices are found to provide sustained controlled release of various compositions to treat 40 the eye without risk of detrimental local and systemic side effects. An object of the present ocular method of delivery is to maximize the amount of drug contained in an intraocular device or implant while minimizing its size in order to prolong the duration of the implant. See, e.g., U.S. Pat. Nos. 45 5,378,475; 6,375,972, and 6,756,058 and U.S. Publications 20050096290 and 200501269448. Such implants may be biodegradable and/or biocompatible implants, or may be nonbiodegradable implants. Biodegradable ocular implants are described, for example, in U.S. Patent Publication No. 50 20050048099. The implants may be permeable or impermeable to the active agent, and may be inserted into a chamber of the eye, such as the anterior or posterior chambers or may be implanted in the schlera, transchoroidal space, or an avascularized region exterior to the vitreous. Alternatively, a contact 55 lens that acts as a depot for compositions of the invention may also be used for drug delivery.

In a preferred embodiment, the implant may be positioned over an avascular region, such as on the sclera, so as to allow for transcleral diffusion of the drug to the desired site of 60 treatment, e.g. the intraocular space and macula of the eye. Furthermore, the site of transcleral diffusion is preferably in proximity to the macula. Examples of implants for delivery of a composition include, but are not limited to, the devices described in U.S. Pat. Nos. 3,416,530; 3,828,777; 4,014,335; 65 4,300,557; 4,327,725; 4,853,224; 4,946,450; 4,997,652; 5,147,647; 5,164,188; 5,178,635; 5,300,114; 5,322,691;

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5,403,901; 5,443,505; 5,466,466; 5,476,511; 5,516,522; 5,632,984; 5,679,666; 5,710,165; 5,725,493; 5,743,274; 5,766,242; 5,766,619; 5,770,592; 5,773,019; 5,824,072; 5,824,073; 5,830,173; 5,836,935; 5,869,079, 5,902,598; 5,904,144; 5,916,584; 6,001,386; 6,074,661; 6,110,485; 6,126,687; 6,146,366; 6,251,090; and 6,299,895, and in WO 01/30323 and WO 01/28474, all of which are incorporated herein by reference.

Examples include, but are not limited to the following: a sustained release drug delivery system comprising an inner reservoir comprising an effective amount of an agent effective in obtaining a desired local or systemic physiological or pharmacological effect, an inner tube impermeable to the passage of the agent, the inner tube having first and second ends and covering at least a portion of the inner reservoir, the inner tube sized and formed of a material so that the inner tube is capable of supporting its own weight, an impermeable member positioned at the inner tube first end, the impermeable member preventing passage of the agent out of the reservoir through the inner tube first end, and a permeable member positioned at the inner tube second end, the permeable member allowing diffusion of the agent out of the reservoir through the inner tube second end; a method for administering a compound of the invention to a segment of an eye, the method comprising the step of implanting a sustained release device to deliver the compound of the invention to the vitreous of the eye or an implantable, sustained release device for administering a compound of the invention to a segment of an eye; a sustained release drug delivery device comprising: a) a drug core comprising a therapeutically effective amount of at least one first agent effective in obtaining a diagnostic effect or effective in obtaining a desired local or systemic physiological or pharmacological effect; b) at least one unitary cup essentially impermeable to the passage of the agent that surrounds and defines an internal compartment to accept the drug core, the unitary cup comprising an open top end with at least one recessed groove around at least some portion of the open top end of the unitary cup; c) a permeable plug which is permeable to the passage of the agent, the permeable plug is positioned at the open top end of the unitary cup wherein the groove interacts with the permeable plug holding it in position and closing the open top end, the permeable plug allowing passage of the agent out of the drug core, through the permeable plug, and out the open top end of the unitary cup; and d) at least one second agent effective in obtaining a diagnostic effect or effective in obtaining a desired local or systemic physiological or pharmacological effect; or a sustained release drug delivery device comprising: an inner core comprising an effective amount of an agent having a desired solubility and a polymer coating layer, the polymer layer being permeable to the agent, wherein the polymer coating layer completely covers the inner core.

Other approaches for ocular delivery include the use of liposomes to target a compound of the present invention to the eye, and preferably to retinal pigment epithelial cells and/or Bruch's membrane. For example, the compound may be complexed with liposomes in the manner described above, and this compound/liposome complex injected into patients with an ocular disease, using intravenous injection to direct the compound to the desired ocular tissue or cell. Directly injecting the liposome complex into the proximity of the retinal pigment epithelial cells or Bruch's membrane can also provide for targeting of the complex with some forms of ocular disease. In a specific embodiment, the compound is administered via intra-ocular sustained delivery (such as VITRA-SERT or ENVISION). In a specific embodiment, the compound is delivered by posterior subtenons injection. In

another specific embodiment, microemulsion particles containing the compositions of the invention are delivered to ocular tissue to take up lipid from Bruch's membrane, retinal pigment epithelial cells, or both.

For optical applications, nanoparticles are a colloidal car- 5 rier system that has been shown to improve the efficacy of the encapsulated drug by prolonging the serum half-life. Polyalkylcyanoacrylates (PACAs) nanoparticles are a polymer colloidal drug delivery system that is in clinical development, as described by Stella et al., J. Pharm. Sci., 2000. 89: p. 10 1452-1464; Brigger et al., Int. J. Pharm., 2001. 214: p. 37-42; Calvo et al., Pharm. Res., 2001. 18: p. 1157-1166; and Li et al., Biol. Pharm. Bull., 2001. 24: p. 662-665. Biodegradable poly(hydroxyl acids), such as the copolymers of poly(lactic acid) (PLA) and poly(lactic-co-glycolide) (PLGA) are being 15 extensively used in biomedical applications and have received FDA approval for certain clinical applications. In addition, PEG-PLGA nanoparticles have many desirable carrier features including (i) that the agent to be encapsulated comprises a reasonably high weight fraction (loading) of the 20 total carrier system; (ii) that the amount of agent used in the first step of the encapsulation process is incorporated into the final carrier (entrapment efficiency) at a reasonably high level; (iii) that the carrier have the ability to be freeze-dried and reconstituted in solution without aggregation; (iv) that the 25 carrier be biodegradable; (v) that the carrier system be of small size; and (vi) that the carrier enhance the particles persistence.

Nanoparticles are synthesized using virtually any biodegradable shell known in the art. In one embodiment, a polymer, such as poly(lactic-acid) (PLA) or poly(lactic-co-glycolic acid) (PLGA) is used. Such polymers are biocompatible and biodegradable, and are subject to modifications that desirably increase the photochemical efficacy and circulation lifetime of the nanoparticle. In one embodiment, the polymer is modified with a terminal carboxylic acid group (COOH) that increases the negative charge of the particle and thus limits the interaction with negatively charge nucleic acid aptamers. Nanoparticles are also modified with polyethylene glycol (PEG), which also increases the half-life and stability of the particles in circulation. Alternatively, the COOH group is converted to an N-hydroxysuccinimide (NHS) ester for covalent conjugation to amine-modified aptamers.

Biocompatible polymers useful in the composition and methods of the invention include, but are not limited to, 45 polyamides, polycarbonates, polyalkylenes, polyalkylene glycols, polyalkylene oxides, polyalkylene terepthalates, polyvinyl alcohols, polyvinyl ethers, polyvinyl esters, polyvinyl halides, polyvinylpyrrolidone, polyglycolides, polysiloxanes, polyurethanes and copolymers thereof, alkyl cellu- 50 lose, hydroxyalkyl celluloses, cellulose ethers, cellulose esters, nitro celluloses, polymers of acrylic and methacrylic esters, methyl cellulose, ethyl cellulose, hydroxypropyl cellulose, hydroxy-propyl methyl cellulose, hydroxybutyl methyl cellulose, cellulose acetate, cellulose propionate, cel- 55 lulose acetate butyrate, cellulose acetate phthalate, carboxylethyl cellulose, cellulose triacetate, cellulose sulphate sodium salt, poly(methyl methacrylate), poly(ethylmethacrylate), poly(butylmethacrylate), poly(isobutylmethacrylate), poly(hexlmethacrylate), poly(isodecylmethacrylate), poly 60 (lauryl methacrylate), poly(phenyl methacrylate), poly(methyl acrylate), poly(isopropyl acrylate), poly(isobutyl acrylate), poly(octadecyl acrylate), polyethylene, polypropylene poly(ethylene glycol), poly(ethylene oxide), poly(ethylene terephthalate), poly(vinyl alcohols), poly(vinyl acetate, poly vinyl chloride polystyrene, polyvinylpryrrolidone, polyhyaluronic acids, casein, gelatin, glutin, polyanhydrides, poly54

acrylic acid, alginate, chitosan, poly(methyl methacrylates), poly(ethyl methacrylates), poly(butylmethacrylate), poly (isobutylmethacrylate), poly(hexlmethacrylate), poly(isodecyl methacrylate), poly(lauryl methacrylate), poly(phenyl methacrylate), poly(methyl acrylate), poly(isopropyl acrylate), poly(isobutyl acrylate), poly(octadecyl acrylate) and combinations of any of these. In one embodiment, the nanoparticles of the invention include PEG-PLGA polymers.

Compositions of the invention may also be delivered topically. For topical delivery, the compositions are provided in any pharmaceutically acceptable excipient that is approved for ocular delivery. Preferably, the composition is delivered in drop form to the surface of the eye. For some application, the delivery of the composition relies on the diffusion of the compounds through the cornea to the interior of the eye.

Those of skill in the art will recognize that the best treatment regimens for using compounds of the present invention to treat an ocular disease can be straightforwardly determined. This is not a question of experimentation, but rather one of optimization, which is routinely conducted in the medical arts. In vivo studies in nude mice often provide a starting point from which to begin to optimize the dosage and delivery regimes. The frequency of injection will initially be once a week, as has been done in some mice studies. However, this frequency might be optimally adjusted from one day to every two weeks to monthly, depending upon the results obtained from the initial clinical trials and the needs of a particular patient.

Human dosage amounts can initially be determined by extrapolating from the amount of compound used in mice, as a skilled artisan recognizes it is routine in the art to modify the dosage for humans compared to animal models. In certain embodiments it is envisioned that the dosage may vary from between about 1 mg compound/Kg body weight to about 5000 mg compound/Kg body weight; or from about 5 mg/Kg body weight to about 4000 mg/Kg body weight or from about 10 mg/Kg body weight to about 3000 mg/Kg body weight; or from about 50 mg/Kg body weight to about 2000 mg/Kg body weight; or from about 100 mg/Kg body weight to about 1000 mg/Kg body weight; or from about 150 mg/Kg body weight to about 500 mg/Kg body weight. In other embodiments this dose may be about 1, 5, 10, 25, 50, 75, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600, 650, 700, 750, 800, 850, 900, 950, 1000, 1050, 1100, 1150, 1200, 1250, 1300, 1350, 1400, 1450, 1500, 1600, 1700, 1800, 1900, 2000, 2500, 3000, 3500, 4000, 4500, 5000 mg/Kg body weight. In other embodiments, it is envisaged that higher doses may be used, such doses may be in the range of about 5 mg compound/Kg body to about 20 mg compound/Kg body. In other embodiments the doses may be about 8, 10, 12, 14, 16 or 18 mg/Kg body weight. Of course, this dosage amount may be adjusted upward or downward, as is routinely done in such treatment protocols, depending on the results of the initial clinical trials and the needs of a particular patient.

### Combination Therapies

Optionally, an angiogenic modulating therapeutic as described herein may be administered in combination with any other standard active angiogenic modulating therapeutics; such methods are known to the skilled artisan and described in *Remington's Pharmaceutical Sciences* by E. W. Martin. For example, an anti-angiogenic peptide of the invention may be administered in combination with any other anti-angiogenic peptide, or with known anti-angiogenic agent. Such agents are listed below (Folkman, *Annu Rev Med.* 57:1-18, 2006).

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Agent	Clinical Trials
1. Alphastatin	
2. Angiostatin	
3. Arresten	
4. Anti-thrombin III (truncated)	
<ol><li>Canstatin</li></ol>	
6. Endostatin	Phase II
7. Fibulin-5	
<ol><li>Fragment of histidine-rich glycoprotein</li></ol>	
9. Interferon-β	Phase III
10. Maspin	
11. 2-methoxyestradiol	Phase II
12. PEX	
<ol><li>Pigment epithelial-derived factor (PEDF)</li></ol>	
<ol><li>Platelet factor 4 (PF4)</li></ol>	
15. Semaphorin 3F	
16. sFlt-1	
17. Tetrahydrocortisol	Phase III
18. Thrombospondin-1 (and -2)	Phase II
19. TIMP-2	
20. Troponin I	
21. Tumstatin	
22. Vasostatin	

For the treatment of a neoplasia, a peptide of the invention is administered in combination with any conventional treatment (e.g., chemotherapy, radiotherapy, hormonal therapy, 25 surgery, cryosurgery). A pharmaceutical composition of the invention may, if desired, include one or more chemotherapeutics typically used in the treatment of a neoplasm, such as abiraterone acetate, altretamine, anhydrovinblastine, auristatin, bexarotene, bicalutamide, BMS184476, 2,3,4,5,6-pen-30 tafluoro-N-(3-fluoro-4-methoxyphenyl)benzene sulfonamide, bleomycin, N,N-dimethyl-L-valyl-L-valyl-N-methyl-L-valyl-L-proly-1-Lproline-t-butylamide, cachectin, cemadotin, chlorambucil, cyclophosphamide, 3',4'-didehydro-4'-deoxy-8'-norvin-caleukoblastine, docetaxol, dox- 35 etaxel, cyclophosphamide, carboplatin, carmustine (BCNU), cisplatin, cryptophycin, cyclophosphamide, cytarabine, dacarbazine (DTIC), dactinomycin, daunorubicin, dolastatin, doxorubicin (adriamycin), etoposide, 5-fluorouracil, finasteride, flutamide, hydroxyurea and hydroxyureataxanes, ifos-40 famide, liarozole, lonidamine, lomustine (CCNU), mechlorethamine (nitrogen mustard), melphalan, mivobulin isethionate, rhizoxin, sertenef, streptozocin, mitomycin, methotrexate, 5-fluorouracil, nilutamide, onapristone, paclitaxel, prednimustine, procarbazine, RPR109881, stramustine 45 phosphate, tamoxifen, tasonermin, taxol, thalidomide, tretinoin, vinblastine, vincristine, vindesine sulfate, and vinflunine. Other examples of chemotherapeutic agents can be found in Cancer Principles and Practice of Oncology by V. T. Devita and S. Hellman (editors), 6th edition (Feb. 15, 2001), 50 Lippincott Williams & Wilkins Publishers. Kits

The invention provides kits for the treatment or prevention of diseases or disorders characterized by excess or undesirable angiogenesis. In one embodiment, the kit includes a 55 therapeutic or prophylactic composition containing an effective amount of one or more peptides described herein in unit dosage form. In some embodiments, the kit comprises a sterile container that contains a therapeutic or prophylactic vaccine; such containers can be boxes, ampules, bottles, vials, 60 tubes, bags, pouches, blister-packs, or other suitable container forms known in the art. Such containers can be made of plastic, glass, laminated paper, metal foil, or other materials suitable for holding medicaments.

If desired a peptide of the invention is provided together 65 with instructions for administering it to a subject having or at risk of developing excess or undesired angiogenesis. The

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instructions will generally include information about the use of the composition for the treatment or prevention of ischemia or for enhancing angiogenesis to a tissue in need thereof. In other embodiments, the instructions include at least one of the following: description of the expression vector; dosage schedule and administration for treatment or prevention of ischemia or symptoms thereof; precautions; warnings; indications; counter-indications; overdosage information; adverse reactions; animal pharmacology; clinical studies; and/or references. The instructions may be printed directly on the container (when present), or as a label applied to the container, or as a separate sheet, pamphlet, card, or folder supplied in or with the container.

### METHODS OF THE INVENTION

The practice of the present invention employs, unless otherwise indicated, conventional techniques of molecular biol-20 ogy (including recombinant techniques), microbiology, cell biology, biochemistry and immunology, which are well within the purview of the skilled artisan. Such techniques are explained fully in the literature, such as, "Molecular Cloning: A Laboratory Manual", second edition (Sambrook, 1989); "Oligonucleotide Synthesis" (Gait, 1984); "Animal Cell Culture" (Freshney, 1987); "Handbook of Experimental Immunology" (Weir, 1996); "Gene Transfer Vectors for Mammalian Cells" (Miller and Calos, 1987); "Current Protocols in Molecular Biology" (Ausubel, 1987); "PCR: The Polymerase Chain Reaction", (Mullis, 1994); "Current Protocols in Immunology" (Coligan, 1991). These techniques are applicable to the production of the polynucleotides and polypeptides of the invention, and, as such, may be considered in making and practicing the invention. Particularly useful techniques for particular embodiments will be discussed in the sections that follow.

### **EXAMPLES**

### Example 1

### Analysis of Peptide Motifs

Using bioinformatic analysis 156 peptides with anti-angiogenic properties were identified based on their sequence similarity with known anti-angiogenic peptides. A number of these peptides were screened for anti-angiogenic activity using an endothelial cell proliferation assay to identify peptide motifs associated with anti-angiogenic activity. Multiple sequence alignments were used to identify peptides having conserved motifs that are common in a variety of sequences. Multiple sequence alignment was performed using the ClustalW algorithm to align sequences of peptides that belong to different protein families including type I thrombospondin repeat-containing proteins, C-X-C chemokines, collagen type IV, somatotropins and serpins. In order to perform the alignment a critical number of peptide sequences were required. The motifs were represented using the single letter abbreviations of the amino acids that are common and the letter "X" to denote a non-common amino acid that intervenes the common letters. If there is more than one non-common amino acid in between, the letter "X" followed by the number of the non-common amino acids was used. For example if there are three non-common amino acids between two conserved letters, we notify it as "a-X3-b", where a and b is the conserved motif. This notation is commonly used to represent motifs.

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Initially multiple sequence alignments to the experimentally tested peptides were performed. The calculation was generalized to all the theoretically predicted fragments. To determine whether the motifs calculated for the experimentally tested fragments were conserved and reproduced in all of 5 the anti-angiogenic predictions. The results obtained were organized by protein family. As described in more detail below, general peptide motifs associated with anti-angiogenic activity were identified for three families of human proteins: Type I thrombospondin (TSP) domain containing 10 proteins, CXC chemokines, and collagens. Using these motifs, 2286 peptides each containing one of the identified motifs were identified in 1977 different proteins present in the human proteome (166 peptides from 54 different proteins listed in Table 2; 1337 peptides from 1170 proteins listed in 15 Table 4; 24 peptides from 24 proteins listed in Table 5; 306 peptides from 288 proteins listed in Table 6; 139 peptides from 139 proteins listed in Table 8; and 314 peptides from 302 different proteins listed in Table 9.

In addition, 12 novel peptide sequences from the Soma- 20 AA#11AA#12AA#13AA#14AA#15AA#16AA#17AA#18AA#19AA#20 totropin, Serpin, and Type IV Collagen families obtained based on the similarity criteria with known anti-angiogenic peptides are listed in Tables 7A, 7B, and 10, respectively.

### Example 2

### Thrombospondin-1 (TSP-1) Repeat-Containing Proteins Derived Peptides

From the 31 predicted and experimentally tested TSP-1 30 containing short peptides 29 share a global 4 letter common motif which is the X2-W-X2-C-X3-C-X2-G-X7 (SEQ ID NO: 2353), or W-X2-C-X3-C-X2-G (SEQ ID NO: 2287) after removing the uncommon edges, resulting in the generic TSP-1 containing 20-mer (FIG. 1). The first amino acid that 35 were identified from the sequences of the peptide fragments succeeds the first cysteine of the motif, or the seventh amino acid of the sequence can alternate between T, S and N. Thus a more generic description of this motif is X2-W-X2-C-(T/S/ N)-X2-C-X2-G-X7 (SEQ ID NO: 2354) with threonine or serine the most abundant alteration for the seventh amino acid 40 position.

By altering the threshold of the conserved amino acids that are common among the sequences of the predicted peptides we can create subsets of peptide families with individual common motifs of greater length than the global 4-letter 45 motif. The threshold here is defined as the percentage of the peptides that share a common motif. Such a subgroup of peptides is one that consists of 18 TSP-1 containing predictions (threshold 60%) that share a seven amino acid long common motif. The motif is the X2-W-X2-C-S-X2-C-G-X1- 50 G-X3-R-X3 (SEQ ID NO: 2355). A common alteration occurs in the 19<sup>th</sup> amino acid, which can be either an arginine or a valine with arginine the most abundant amino acid. In that case the motif is written X2-W-X2-C-S-X2-C-G-X1-G-X3-R-X1-(R/V)-X1 (SEQ ID NO: 2387). Similarly the ninth 55 amino acid can be altered by either arginine, serine or threonine. In that case the motif can be represented as X2-W-X2-C-S-X1-(S/R/T)-C-G-X1-G-X3-R-X1-(R/V)-X1 (SEQ ID NO: 2391) with threonine the most abundant amino acid (FIG. 2A). Similarly another motif with 45% threshold, com- 60 mon in 13 sequences, is the 5 letter motif X1-P-W-X2-C-X3-C-X2-G-X7 (SEQ ID NO: 2407). The common alterations of this motif can be described as (S/G/Q)-P-W-X2-C-(T/S)-X2-C-(G/S)-X1-G-X3-(R/S)-X3 (FIG. 2B) (SEQ ID NO: 2417).

In addition to calculating the motifs that are present within 65 the sequences of the predicted fragments one can analyze all the possible amino acids that are present within the 29 peptide

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sequences from which the motifs were calculated. This 20-mer with all the possible substitutions is presented in Table 1 along with the frequencies that each amino acid is present in the 29 sequences.

TABLE 1 The TSP-1 containing 20-mer with

	all	the p	ossib	le am	ino a	cid s	ubsti	tution	ıs
A#1	AA#2	AA#3	AA#4	AA#5	AA#6	AA#7	. ~		: 2420 AA#10
(9)				A(5)			A(6)	T(15) S(10)	

$\begin{array}{cccccccccccccccccccccccccccccccccccc$							
A(1) R(1) T(2)	5	T(9) G(6) Q(2)	E(5) S(3) A(2) Q(1)	T(5) G(5) E(2) D(1)	A(5) Q(4) D(3) E(3)	N(2) A(6) T(1) R(5) K(4) G(2)	S(10) R(3)
		A(1)		R(1)	K(1) R(1)	S(2) T(2)	

G(26	)G(10)G(2	29)V(8)	Q(11)	T(10	R(26)	S(5)	R(15)R(1)
S(2)	K(4)	I(4)	S(7)	F(4)	S(2)	T(5)	V(1)
N(1)	R(4)	M(3)	R(6)	K(3)	Q(1)	V(5)	
	M(4)	T(3)	K(2)	Q(3)		R(3)	
	T(2)	H(2)	Y(2)	S(3)		H(3)	
	L(2)	A(1)	A(1)	L(2)		E(2)	
	D(1)	E(1)		E(1)		Q(2)	
	S(1)	F(1)		M(1)		A(1)	
	P(1)	K(1)		N(1)		I(1)	
		R(1)		V(1)			
		S(1)					
		Q(1)					
		W(1)					
		Y(1)					

The above motifs, for both the TSP-1 containing proteins that have already been experimentally tested in proliferation assay. The specific approach for identification of motifs within groups of sequences can be generalized for the case of all the theoretically predicted anti-angiogenic fragments. For the TSP-1 repeat-containing protein derived fragments the multiple sequence alignment calculations are repeated, but now all of the theoretically predicted fragments are included. The same approach is also utilized for the case of collagens where only the experimentally tested pool of sequences is not sufficient to yield statistically significant results. In that case after including all the theoretically predicted fragments we are able to identify common motifs.

For the cases of all the theoretically predicted TSP-1 containing proteins, multiple sequence alignment yields a common motif within 97% of all the tested sequences. This motif is the already identified W-X2-C-X3-C-X2-G (SEQ ID NO: 2287) (FIG. 3) and a generic 20-mer can be expressed as X2-W-X2-C-X3-C-X2-G-X7 (SEQ ID NO: 2353). It is interesting that this motif is not a characteristic of only the TSP-1 domains, in other words in not a signature for TSP-1. When its presence was tested for all the TSP-1 containing proteins it was identified only within a subset of this family. Moreover, it is present within the type-2 thrombospondin containing proteins (TSP-2), which have already been shown to be associated with anti-angiogenic activity. In other words we claim that the motif W-X2-C-X3-C-X2-G (SEQ ID NO: 2287), although present within a large portion of the TSP-1 containing proteins, is not a signature for a generic TSP-1 containing protein but only for those proteins with putative anti-angiogenic activity that may or may not belong to the specific protein family. Moreover, as observed within the sequences of the experimentally tested fragments and is also reproduced

Accession

Number|Protein Name

in the case of all the theoretically predicted fragments, the amino acid following the first cysteine of the motif can alternate between T, S and N. Thus a more specific description of the motif is the W-X2-C-(T/S/N)-X2-C-X2-G (SEQ ID NO: 2421) with serine and threonine being the predominant amino 5 acids in the position following the first cysteine.

A common alteration occurs in the 19<sup>th</sup> amino acid of the 20-mer which can be either an arginine or a valine with arginine the most abundant amino acid. In that case the motif is written X2-W-X2-C-(T/S/N)-X2-C-X2-G-X5-(R/V)-X <sup>10</sup> (SEQ ID NO: 2427).

The most generic 4-common letter motif identified within the peptide sequences is W-X2-C-X3-C-X2-G (SEQ ID NO: 2287). The ScanProsite tool can be used to search the human proteome Prosite database at the Swiss Institute of Bioinformatics. Using the aforementioned motif as a query identified this motif in 166 locations of 54 different proteins listed in Table 2 (SEQ ID Nos. 1-166).

TABLE 2

TSPs Motif: W-X(2)-C-X(3)-C-X(2)-G (SEQ ID NO: 2287) Number of Locations: 166

Number of Different Proteins: 54

	rumber of Biner	chi i rotch	10. 5 .		25
#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence	
1	O00622 CYR61 HUMAN	236	246	WsqCsktCgtG	•
2	O14514 BAI1_HUMAN	270	280	WgeCtrdCggG	30
3	O14514 BAI1_HUMAN	363	373	WsvCsstCgeG	50
4	O14514 BAI1_HUMAN	418	428	WslCsstCgrG	
5	O14514 BAI1_HUMAN	476	486	WsaCsasCsqG	
6	O14514 BAI1_HUMAN	531	541	WgsCsvtCgaG	
7	O15072 ATS3_HUMAN	975	985	WseCsvtCgeG	
8	O60241 BAI2_HUMAN	306	316	WsvCsltCgqG	2.5
9	O60241 BAI2_HUMAN	361	371	WslCsrsCgrG	35
10	O60241 BAI2_HUMAN	416	426	WgpCstsCanG	
11	O60241 BAI2_HUMAN	472	482	WslCsktCdtG	
12	O60242 BAI3_HUMAN	300	310	WstCsvtCgqG	
13	O60242 BAI3 HUMAN	354	364	WslCsftCgrG	
14	O60242 BAI3_HUMAN	409	419	WsqCsvtCsnG	
15	O60242 BAI3 HUMAN	464	474	WsgCsksCdgG	40
16	O75173 ATS4_HUMAN	529	539	WgdCsrtCggG	
17	O76076 WISP2 HUMAN	201	211	WgpCsttCglG	
18	O95185 UNC5C_HUMAN	269	279	WsvCnsrCgrG	
19	O95388 WISP1 HUMAN	223	233	WspCstsCglG	
20	O95389 WISP3 HUMAN	216	226	WtpCsrtCgmG	
21	O95450 ATS2 HUMAN	863	873	WspCskpCggG	45
22	O95450 ATS2_HUMAN	984	994	WsqCsvtCgnG	
23	P07996 TSP1 HUMAN	388	398	WtsCstsCgnG	
24	P07996 TSP1 HUMAN	444	454	WssCsvtCgdG	
25	P07996 TSP1 HUMAN	501	511	WdiCsvtCggG	
26	P13671 CO6_HUMAN	32	42	WtsCsktCnsG	
27	P13671 CO6 HUMAN	75	85	WgrCpinCllG	50
28	P14222 PERF_HUMAN	374	384	WrdCsrpCppG	50
29	P27918 PROP_HUMAN	86	96	WapCsvtCseG	
30	P27918 PROP_HUMAN	145	155	WepCsvtCskG	
31	P27918 PROP HUMAN	202	212	WtpCsasChgG	
32	P29279 CTGF_HUMAN	206	216	WsaCsktCgmG	
33	P35442 TSP2 HUMAN	390	400	WtqCsvtCgsG	
34	P35442 TSP2 HUMAN	446	456	WssCsvtCgvG	55
35	P35442 TSP2 HUMAN	503	513	WsaCtvtCagG	
36	P48745 NOV_HUMAN	213	223	WtaCsksCgmG	
37	P49327 FAS HUMAN	627	637	WeeCkgrCppG	
38	P58397 ATS12_HUMAN	551	561	WshCsrtCgaG	
39	P58397 ATS12_HUMAN	832	842	WteCsvtCgtG	
40	P58397 ATS12_HUMAN	952	962	WseCsvsCggG	60
41	P58397 ATS12_HUMAN	1321	1331	WseCsttCglG	
42	P58397 ATS12_HUMAN	1372	1382	WskCsmCsgG	
43	P58397 ATS12_HUMAN	1431	1441	WsqCsrsCggG	
44	P58397 ATS12_HUMAN	1479	1489	WdlCstsCggG	
45	P59510 ATS20 HUMAN	976	986	WsqCsrsCggG	
46	P59510 ATS20_HUMAN	1031	1041	WseClvtCgkG	65
47	P59510 ATS20_HUMAN	1031	1041	WgpCtttCghG	
47	1 33310 ALBZU_HUIVIAIN	1000	1090	** gpCtttCgIIG	

### TABLE 2-continued

TSPs Motif: W-X(2)-C-X(3)-C-X(2)-G (SEQ ID NO: 2287) Number of Locations: 166 Number of Different Proteins: 54

First

Amino

acid

Last

Amino

acid Sequence

10	48	P59510 ATS20_HUMAN	1162	1172	WtpCsvsCgrG
	49	P59510 ATS20_HUMAN	1217	1227	WspCsasCghG
	50	P59510 ATS20_HUMAN	1314	1324	WgsCsssCsgG
	51	P59510 ATS20_HUMAN	1368	1378	WgeCsqtCggG
	52	P59510 ATS20_HUMAN	1427	1437	WtsCsasCgkG
	53	P59510 ATS20_HUMAN	1483	1493	WneCsvtCgsG
15	54	P59510 ATS20_HUMAN	1664	1674	WskCsvtCgiG
13	55	P82987 ATL3_HUMAN	84	94	WsdCsrtCggG
	56	P82987 ATL3_HUMAN	427	437	WtaCsvsCggG
	57	P82987 ATL3_HUMAN	487	497	WsqCtvtCgrG
	58	P82987 ATL3_HUMAN	573	583	WsaCsttCgpG
	59	P82987 ATL3_HUMAN	712	722	WgpCsatCgvG
20	60	P82987 ATL3_HUMAN	768	778	WqqCsrtCggG
20	61	P82987 ATL3_HUMAN	828	838	WskCsvsCgvG
	62	P82987 ATL3_HUMAN	1492	1502	WsqCsvsCgeG
	63 64	P82987 ATL3_HUMAN	1606	1616	WkpCtaaCgrG
	65	Q13591 SEM5A_HUMAN	604 662	614 672	WspCsttCgiG
	66	Q13591 SEM5A_HUMAN Q13591 SEM5A_HUMAN	793	803	WerCtaqCggG WsqCsrdCsrG
25	67	Q13591 SEM5A_HUMAN	850	860	WtkCsatCggG
20	68	Q496M8 CI094_HUMAN	259	269	WaCtrsCggG
	69	Q6S8J7 POTE8_HUMAN	27	37	WccCcfpCcrG
	70	Q6UXZ4 UNC5D_HUMAN	261	271	WsaCnvrCgrG
	71	Q6UY14 ATL4 HUMAN	53	63	WasCsqpCgvG
	72	Q6UY14 ATL4 HUMAN	732	742	WtsCsrsCgpG
30	73	Q6UY14 ATL4_HUMAN	792	802	WsqCsvrCgrG
-	74	Q6UY14 ATL4_HUMAN	919	929	WgeCsseCgsG
	75	Q6UY14 ATL4_HUMAN	979	989	WspCsrsCqgG
	76	Q6ZMM2 ATL5_HUMAN	44	54	WtrCsssCgrG
	77	Q76LX8 ATS13_HUMAN	1081	1091	WmeCsvsCgdG
	78	Q86TH1 ATL2_HUMAN	56	66	WtaCsrsCggG
35	79	Q86TH1 ATL2_HUMAN	631	641	WseCsrtCgeG
	80	Q86TH1 ATL2_HUMAN	746	756	WgpCsgsCgqG
	81	Q86TH1 ATL2_HUMAN	803	813	WerCnttCgrG
	82	Q86TH1 ATL2_HUMAN	862	872	WseCtktCgvG
	83	Q8IUL8 CILP2_HUMAN	155	165	WgpCsgsCgpG
	84	Q8IZJ1 UNC5B_HUMAN	255	265	WspCsnrCgrG
40	85	Q8N6G6 ATL1_HUMAN	42	52	WseCsrtCggG
	86 87	Q8N6G6 ATL1_HUMAN	385 445	395 455	WtaCsssCggG
	88	Q8N6G6 ATL1_HUMAN Q8TE56 ATS17_HUMAN	552	562	WspCtvtCgqG WsmCsrtCgtG
	89	Q8TE56 ATS17_HUMAN	809	819	WegCsvqCggG
	90	Q8TE56 ATS17_HUMAN	870	880	WspCsatCekG
	91	Q8TE56 ATS17_HUMAN	930	940	WsqCsasCgkG
45	92	Q8TE56 ATS17_HUMAN	981	991	WstCsstCgkG
	93	Q8TE57 ATS16_HUMAN	595	605	WspCsrtCggG
	94	Q8TE57 ATS16_HUMAN	936	946	WsaCsrtCggG
	95	Q8TE57 ATS16_HUMAN	995	1005	WaeCshtCgkG
	96	Q8TE57 ATS16_HUMAN	1060	1070	WsqCsvtCerG
	97	Q8TE57 ATS16_HUMAN	1135	1145	WsqCtasCggG
50	98	Q8TE58 ATS15_HUMAN	848	858	WgpCsasCgsG
	99	Q8TE58 ATS15_HUMAN	902	912	WspCsksCgrG
	100	Q8TE59 ATS19_HUMAN	642	652	WspCsrtCsaG
	101	Q8TE59 ATS19_HUMAN	924	934	WedCdatCggG
	102	Q8TE59 ATS19_HUMAN	985	995	WtpCsrtCgkG
	103	Q8TE59 ATS19_HUMAN	1096	1106	WskCsitCgkG
55	104 105	Q8TE60 ATS18_HUMAN	598 940	608 950	WseCsrtCggG
	106	Q8TE60 ATS18_HUMAN Q8TE60 ATS18_HUMAN	1000	1010	WstCskaCagG WsqCsktCgrG
	107	Q8TE60 ATS18_HUMAN	1061	1071	WsqCsatCglG
	108	Q8TE60 ATS18_HUMAN	1132	1142	WqqCtvtCggG
	109	Q8WXS8 ATS14_HUMAN	856	866	WapCskaCggG
	110	Q8WXS8 ATS14_HUMAN	977	987	WsqCsatCgeG
60	111	Q92947 GCDH HUMAN	225	235	WarCedgCirG
	112	Q96RW7 HMCN1_HUMAN	4538	4548	WraCsvtCgkG
	113	Q96RW7 HMCN1_HUMAN	4595	4605	WeeCtrsCgrG
	114	Q96RW7 HMCN1_HUMAN	4652	4662	WgtCsesCgkG
	115	Q96RW7 HMCN1_HUMAN	4709	4719	WsaCsvsCggG
	116	Q96RW7 HMCN1_HUMAN	4766	4776	WgtCsrtCngG
65	117	Q96RW7 HMCN1_HUMAN	4823	4833	WsqCsasCggG
	118	Q99732 LITAF_HUMAN	116	126	WlsCgslCllG

55

61

TABLE 2-continued

## **62** TABLE 2-continued

TSPs		
Motif: W-X(2)-C-X(3)-C-X(2)-G (SEQ ID NO: 2287)		
Number of Locations: 166	_	
Number of Different Proteins: 54	5	

TSPs
Motif: W-X(2)-C-X(3)-C-X(2)-G (SEQ ID NO: 2287)
Number of Locations: 166
Number of Different Proteins: 54

		First	Last	
	Accession	Amino	Amino	_
#	Number Protein Name	acid	acid	Sequence
119	Q9C0I4 THS7B_HUMAN	49	59	WgrCtgdCgpG
120	Q9C0I4 THS7B_HUMAN	345	355	WspCsktCrsG
121	Q9C0I4 THS7B_HUMAN	746	756	WtpCprmCqaG
122	Q9C0I4 THS7B_HUMAN	1009	1019	WgsCsssCgiG
123	Q9C0I4 THS7B_HUMAN	1258	1268	WteCsqtCghG
124	Q9C0I4 THS7B_HUMAN	1381	1391	WstCeltCidG
125	Q9H324 ATS10_HUMAN	530	540	WgdCsrtCggG
126	Q9H324 ATS10_HUMAN	808	818	WtkCsaqCagG
127	Q9H324 ATS10_HUMAN	867	877	WslCsrsCdaG
128	Q9H324 ATS10_HUMAN	927	937	WseCtpsCgpG
129	Q9H324 ATS10_HUMAN	986	996	WgeCsaqCgvG
130	Q9HCB6 SPON1_HUMAN	510	520	WspCsisCgmG
131	Q9HCB6 SPON1_HUMAN	567	577	WdeCsatCgmG
132	Q9HCB6 SPON1_HUMAN	623	633	WsdCsvtCgkG
133	Q9HCB6 SPON1_HUMAN	677	687	WseCnksCgkG
134	Q9HCB6 SPON1_HUMAN	763	773	WseCtklCggG
135	Q9NS62 THSD1_HUMAN	349	359	WsqCsatCgdG
136	Q9P283 SEM5B_HUMAN	615	625	WalCstsCgiG
137	Q9P283 SEM5B_HUMAN	673	683	WskCssnCggG
138	Q9P283 SEM5B_HUMAN	804	814	WssCsrdCelG
139	Q9P283 SEM5B_HUMAN	861	871	WspCsasCggG
140	Q9P2N4 ATS9_HUMAN	1006	1016	WteCsksCdgG
141	Q9P2N4 ATS9_HUMAN	1061	1071	WseClvtCgkG
142	Q9P2N4 ATS9_HUMAN	1116	1126	WvqCsvtCgqG
143	Q9P2N4 ATS9_HUMAN	1191	1201	WtpCsatCgkG
144	Q9P2N4 ATS9_HUMAN	1247	1257	WssCsvtCgqG
145	Q9P2N4 ATS9_HUMAN	1337	1347	WgaCsstCagG
146	Q9P2N4 ATS9_HUMAN	1391	1401	WgeCtklCggG
147	Q9P2N4 ATS9_HUMAN	1450	1460	WssCsvsCgrG
148	Q9P2N4 ATS9_HUMAN	1506	1516	WsqCsvsCgrG
149	Q9P2N4 ATS9_HUMAN	1564	1574	WqeCtktCgeG
150	Q9P2N4 ATS9_HUMAN	1621	1631	WseCsvtCgkG

First Last Accession Amino Amino Sequence Number|Protein Name acid acid Q9UHI8|ATS1\_HUMAN WgeCsksCelG 153 863 10 Q9UHI8|ATS1\_HUMAN 917 927 WssCsktCgkG Q9UKP4|ATS7\_HUMAN WsiCsrsCgmG 547 156 Q9UKP4|ATS7\_HUMAN 924 934 WtkCtvtCgrG Q9UKP5|ATS6\_HUMAN 157 519 529 WgeCsrtCggG Q9UKP5|ATS6\_HUMAN WseCsatCagG 158 801 811 Q9UNA0|ATS5\_HUMAN 576 WgqCsrsCggG 159 586 15 Q9UNA0|ATS5\_HUMAN WlaCsrtCdtG 884 894 160 Q9UP79|ATS8\_HUMAN WgeCsrtCggG 536 546 161 Q9UP79|ATS8\_HUMAN 842 852 WseCsstCgaG 162 Q9UPZ6|THS7A\_HUMAN 203 213 WseCsktCgsG 163 O9UPZ6|THS7A HUMAN 780 WtsCpssCkeG 164 790 O9UPZ6|THS7A HUMAN 1044 1054 WsrCsksCgsG 165 20 Q9UPZ6|THS7A\_HUMAN 1423 1433 WslCqltCvnG 166

These peptides are likely to have anti-angiogenic activity. Methods for testing for such activity are described herein.

#### Example 3

### Peptides Derived from C-X-C Chemokines

For the six predicted and experimentally tested C-X-C chemokines, all of them contain a six amino acid common motif. Following the thus far used notation this motif can be described as X-G-X3-C-L-X-P-X10-K-X-L (SEQ ID NO: 2432) (FIG. 4). There are few common alterations that occur within the sequences of the predicted fragments. For all those cases the motif can be re-written as (N/D)-G-(R/K)-X2-C-L-(N/D)-P-X2-(P/N)-X2-(K/Q)-(K/Q)-(I/V)-(I/V)-(E/Q)-K-X-L (SEQ ID NO: 2436).

TABLE 3

		all		C-X-C c ossible						
AA#1	AA#2	AA#3	AA#4	AA#5	AA#6	AA#7	AA#8	•	EQ ID NO AA#10	

AA#1	AA#2	AA#3	AA#4	AA#5	AA#6	AA#7	AA#8	(SE AA#9	Q ID NO AA#10	D: 2437) AA#11
N(4) D(2)	G(6)	R(3) K(3)	K(3) E(2) Q(1)	A(2) I(2) L(1) V(1)	C(6)	L(6)	D(4) N(2)	P(6)	A(2) E(2) D(1) K(1)	A(3) S(2) E(1)
AA#12	AA#13	AA#14	AA#15	AA#16	AA#17	AA#18	AA#19	AA#20	AA#21	AA#22
P(6)	F(2) I(1) M(1) R(1) W(1)	V(3) L(2) I(1)	K(4) Q(2)	K(5) R(1)	I(3) V(3)	I(4) V(2)	E(3) Q(3)	K(6)	I(3) F(1) K(1) M(1)	L(6)

### TABLE 2-continued

TSPs
Motif: W-X(2)-C-X(3)-C-X(2)-G (SEQ ID NO: 2287)
Number of Locations: 166
Number of Different Participes 54

#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
151	Q9P2N4 ATS9_HUMAN	1686		WgsCsvsCgvG
152	Q9UHI8 ATS1_HUMAN	568		WgdCsrtCggG

The generic 22-mer of the predicted C-X-C chemokines including all the possible substitutions is presented in Table 3.

The case of the motif calculation for the theoretically predicted C-X-C chemokines is extremely interesting as in this calculation both short and long fragments are included. If the motifs that were identified within the experimentally tested short fragments are present in the longer ones as well, this might help localize possible anti-angiogenic activity within the longer fragments.

When repeating the calculations with all the theoretically predicted C-X-C chemokines this reproduced the X-G-X3-C-L-X-P-X10-K-X-L motif (SEQ ID NO: 2432) as predicted

Accession

Number|Protein Name

63

when the motifs were calculated in the experimentally tested short fragments, but with minimal alterations (FIG. 5).

For the case of all the theoretically predicted C-X-C chemokines a more generic 22-mer can be described as (N/D/K)-G-X3-C-L-(D/N)-(P/L)-X5-(K/Q)-(K/R/N)-(I/V/L)-(I/V/L)-X6. From the above analysis it also becomes obvious that we can localize the activity of the longer predicted fragments at the sites where the predominant motif from the experimentally tested peptides resides.

Similarly to the type I thrombospondin containing proteins one can consider the most generic 3-common letter motif that is identified within the peptide sequences: G-X3-C-L, and search for its existence within the proteome and identify novel peptides that may contain it. Using as a query the aforementioned motif we utilize the ScanProsite tool to search the Prosite database at the Swiss Institute of Bioinformatics in order to identify protein location that may contain it. The G-X3-C-L motif is identified in 1337 locations of 1170 proteins listed in Table 4 (SEQ ID Nos. 167-1503).

TABLE 4

### CXCs Motif: G-X(3)-C-L Number of Locations: 1337 Number of Different Proteins: 1170

First Last Accession Amino Amino Number Protein Name acid acid Sequence O00142|KITM\_HUMAN GkttCL O00167|EYA2\_HUMAN 361 366 GanlCL 169 O00220|TR10A\_HUMAN 332 337 GeaqCL O00291|HIP1\_HUMAN 699 704 GattCL O00409|FOXN3\_HUMAN 465 470 GirsCL O00444|PLK4\_HUMAN 775 780 GhriCL 173 O00462|MANBA\_HUMAN 744 749 GeavCL 174 O00468|AGRIN\_HUMAN 1549 1554 GdhpCL 175 O00468|AGRIN\_HUMAN 2012 2017 GfvgCL O00476|NPT4\_HUMAN 144 149 GeveCL 176 177 O00488|ZN593\_HUMAN GlhrCL 41 46 O00501|CLD5 HUMAN 178 10 15 GlvlCL 179 O00624|NPT3 HUMAN 220 225 GeveCL 180 O14514|BAI1 HUMAN 243 248 GpenCL 736 O14522|PTPRT\_HUMAN 741 181 **GtplCL** O14548|COX7R HUMAN 97 102 GtivCL 182 O14617IAP3D1\_HUMAN 1113 GhhvCL. 183 1118 O14628|ZN195 HUMAN 184 51 56 GlitCL O14772IFPGT HUMAN 515 520 GnktCL. 185 186 O14773|TPP1\_HUMAN GlqaCL 261 266 187 O14792|OST1 HUMAN GrdrCL 188 O14817|TSN4 HUMAN 68 73 GfvgCL 189 O14841|OPLA\_HUMAN 1240 1245 GdvfCL 190 O14842|FFAR1 HUMAN 166 171 **GspvCL** 191 O14894|T4S5 HUMAN 100 105 GaivCL 192 O14981|BTAF1 HUMAN 608 613 GawlCL 193 O15021|MAST4\_HUMAN 1534 1539 GsheCL 194 O15031|PLXB2\_HUMAN 308 313 GaglCL 195 O15056|SYNJ2\_HUMAN 27 32 GrddCL 196 O15060|ZBT39\_HUMAN 272 277 GtnsCL 197 O15063|K0355 HUMAN 244 249 GcdgCL 198 O15067|PUR4 HUMAN 914 919 GlvtCL 199 O15067|PUR4\_HUMAN 1040 1045 **GpsyCL** 200 O15084|ANR28\_HUMAN 449 454 GnleCL 201 O15084|ANR28\_HUMAN 549 554 GhrlCL 202 O15084|ANR28\_HUMAN 661 GhseCL O15105|SMAD7\_HUMAN 293 298 203 GngfCL 204 O15146|MUSK\_HUMAN 648 653 GkpmCL 205 O15229|KMO\_HUMAN 320 325 GfedCL 206 O15230|LAMA5\_HUMAN 1933 1938 GrtqCL O15296|LX15B\_HUMAN 157 162 GwphCL 208 O15305|PMM2\_HUMAN 10 GpalCL O15354|GPR37\_HUMAN 448 453 GcyfCL 210 O15379|HDAC3\_HUMAN 214 219 GryyCL 211 O15397|IPO8\_HUMAN 148 153 GsllCL

GkivCL

212 O15554|KCNN4\_HUMAN

64

### TABLE 4-continued

#### CXCs Motif: G-X(3)-C-L Number of Locations: 1337 Number of Different Proteins: 1170

First

Amino

acid

Last

Amino

acid

Sequence

	π	Number Trotem Name	acid	acid	Bequence
10	213	O43156 K0406_HUMAN	642	647	GkdfCL
	214	O43175 SERA_HUMAN	111	116	GmimCL
	215	O43175 SERA_HUMAN	416	421	GfgeCL
	216	O43184 ADA12_HUMAN	407	412	GmgvCL
	217 218	O43283 M3K13_HUMAN	133 32	138 37	GlfgCL
	219	O43396 TXNL1_HUMAN O43396 TXNL1_HUMAN	144	149	GegpCL GfdnCL
15	220	O43405 COCH_HUMAN	10	15	GlgvCL
	221	O43541 SMAD6_HUMAN	363	368	GsgfCL
	222	O43609 SPY1_HUMAN	219	224	GtcmCL
	223	O43638 FREA_HUMAN	315	320	GltpCL
	224	O43747 AP1G1_HUMAN	65	70	GqleCL
20	225 226	O43820 HYAL3_HUMAN O43837 IDH3B_HUMAN	12 181	17 186	GvalCL GvieCL
	227	O43889 CREB3_HUMAN	330	335	GntsCL
	228	O60244 CRSP2_HUMAN	447	452	GnseCL
	229	O60266 ADCY3_HUMAN	44	49	GsclCL
	230	O60266 ADCY3_HUMAN	944	949	GgieCL
25	231	O60292 SI1L3_HUMAN	658	663	GekvCL
23	232 233	O60423 AT8B3_HUMAN O60504 VINEX_HUMAN	238 478	243 483	GdvvCL GehiCL
	234	O60508 PRP17_HUMAN	320	325	GerrCL
	235	O60613 SEP15_HUMAN	4	9	GpsgCL
	236	O60656 UD19_HUMAN	510	515	GyrkCL
	237	O60662 KBTBA_HUMAN	447	452	GmiyCL
30	238	O60669 MOT2_HUMAN	93	98	GllcCL
	239 240	O60673 DPOLZ_HUMAN O60704 TPST2_HUMAN	47 229	52 234	GqktCL GkekCL
	241	O60706 ABCC9_HUMAN	1046	1051	GiflCL
	242	O60883 ETBR2_HUMAN	315	320	GcyfCL
	243	O75037 KI21B_HUMAN	1454	1459	GpvmCL
35	244	O75037 KI21B_HUMAN	1617	1622	GltpCL
	245	O75052 CAPON_HUMAN	420	425	GrrdCL
	246 247	O75077 ADA23_HUMAN O75078 ADA11 HUMAN	487 429	492 434	GggaCL GggsCL
	248	O75094 SLIT3_HUMAN	1428	1433	GepyCL
	249	O75095 MEGF6_HUMAN	695	700	GaclCL
	250	O75173 ATS4_HUMAN	19	24	GaqpCL
40	251	O75173 ATS4_HUMAN	419	424	GyghCL
	252	O75311 GLRA3_HUMAN	387	392	GmgpCL
	253 254	O75326 SEM7A_HUMAN O75342 LX12B_HUMAN	499 299	504 304	GchgCL GegtCL
	255	O75342 LX12B_HUMAN	552	557	GfprCL
	256	O75346 ZN253_HUMAN	131	136	GlnqCL
45	257	O75426 FBX24_HUMAN	119	124	GrrrCL
	258	O75436 VP26A_HUMAN	169	174	GiedCL
	259	O75443 TECTA_HUMAN	1687	1692	GdgyCL
	260 261	O75445 USH2A_HUMAN O75445 USH2A_HUMAN	1668 4401	1673 4406	GfvgCL GqglCL
	262	O75446 SAP30_HUMAN	64	69	GqlcCL
50	263	O75508 CLD11_HUMAN	164	169	GavlCL
	264	O75569 PRKRA_HUMAN	268	273	GqyqCL
	265	O75592 MYCB2_HUMAN	1087	1092	GfgvCL
	266	O75636 FCN3_HUMAN	16	21	GgpaCL
	267 268	O75678 RFPL2_HUMAN O75679 RFPL3_HUMAN	117 56	122 61	GcavCL GctvCL
	269	O75689 CENA1_HUMAN	37	42	GvfiCL
55	270	O75691 UTP20_HUMAN	2132	2137	GalqCL
	271	O75694 NU155_HUMAN	230	235	GkdgCL
	272	O75843 AP1G2_HUMAN	67	72	GqmeCL
	273	O75886 STAM2_HUMAN	42	47	GakdCL
	274 275	O75911 DHRS3_HUMAN O75916 RGS9_HUMAN	168 642	173 647	GhivCL GsgtCL
60	276	O75923 DYSF_HUMAN	378	383	GsgiCL GahfCL
	277	O75923 DYSF_HUMAN	1574	1579	GpqeCL
	278	O75925 PIAS1_HUMAN	431	436	GvdgCL
	279	O75954 TSN9_HUMAN	4	9	GclcCL
	280	O75954 TSN9_HUMAN	68	73	GflgCL
65	281	O76000 OR2B3_HUMAN	108	113	GateCL
00	282 283	O76013 K1H6_HUMAN O76064 RNF8_HUMAN	58 15	63 20	GlgsCL GrswCL
	283	O/0004 KINF6_HUMAN	13	20	GISWCL

TABLE 4-continued

#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence		#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
	O76075 DFFB_HUMAN O94759 TRPM2_HUMAN	43 272	48 277	GsrlCL GnltCL	10		P02788 TRFL_HUMAN P02788 TRFL_HUMAN	213 549	218 554	GafkCL GafrCL
	O94759 TRPM2_HUMAN	713	718	GkttCL			P03986 TCC_HUMAN	28	33	GtylCL
	O94761 RECQ4_HUMAN	543	548	GlppCL			P04350 TBB4 HUMAN	235	240	GvttCL
	O94779 CNTN5_HUMAN	169	174	GhyqCL			P04920 B3A2_HUMAN	751	756	GvvfCL
289	O94779 CNTN5_HUMAN	265	270	GsyiCL		360	P05108 CP11A_HUMAN	458	463	GvrqCL
290	O94779 CNTN5_HUMAN	454	459	GmyqCL	15	361	P05141 ADT2_HUMAN	155	160	GlgdCL
	O94829 IPO13_HUMAN	159	164	GqgrCL	13	362	P05549 AP2A_HUMAN	371	376	GiqsCL
	O94856 NFASC_HUMAN	312	317	GeyfCL			P06401 PRGR_HUMAN	484	489	GasgCL
	O94887 FARP2_HUMAN	192	197	GqqhCL			P06756 ITAV_HUMAN	905	910	GvaqCL
	O94900 TOX_HUMAN	22	27	GpspCL			P07202 PERT_HUMAN	819	824	GgfqCL
	O94907 DKK1_HUMAN	107	112	GvqiCL			P07339 CATD_HUMAN	362	367	GktlCL
	O94919 ENDD1_HUMAN O94933 SLIK3_HUMAN	371 898	376 903	GiesCL GfvdCL	20		P07357 CO8A_HUMAN P07437 TBB5_HUMAN	117 235	122 240	GdqdCL GvttCL
	O94955 RHBT3_HUMAN	386	391	GkinCL			P07686 HEXB_HUMAN	483	488	GgeaCL
	O94956 SO2B1_HUMAN	449	454	GmllCL			P07814 SYEP_HUMAN	261	266	GhscCL
	O95071 EDD1_HUMAN	531	536	GtqvCL			P07942 LAMB1_HUMAN	1052	1057	GqclCL
	O95153 RIMB1_HUMAN	79	84	GaeaCL			P07988 PSPB_HUMAN	244	249	GicqCL
302	O95153 RIMB1_HUMAN	1485	1490	GlasCL		373	P08151 GLI1_HUMAN	14	19	GepcCL
303	O95163 IKAP_HUMAN	472	477	GfkvCL	25		P08151 GLI1_HUMAN	828	833	GlapCL
	O95202 LETM1_HUMAN	43	48	GlrnCL			P08243 ASNS_HUMAN	8	13	GsddCL
	O95210 GET1_HUMAN	285	290	GdheCL			P08319 ADH4_HUMAN	241	246	GatdCL
	O95239 KIF4A_HUMAN	27	32	GcqmCL		377	_	212	217	GafrCL
	O95248 MTMR5_HUMAN O95248 MTMR5_HUMAN	159 381	164 386	GlnvCL		378	P08582 TRFM_HUMAN	558 424	563 429	GafrCL GarvCL
	O95255 MRP6_HUMAN	845	850	GyrwCL GalvCL	30	380	_	139	144	Garvel
	O95255 MRP6_HUMAN	943	948	GtplCL	30		P08709 FA7_HUMAN	14	19	GlqgCL
	O95255 MRP6_HUMAN	992	997	GllgCL			P08922 ROS HUMAN	2248	2253	GdviCL
	O95256 I18RA_HUMAN	447	452	GyslCL		383	P09001 RM03_HUMAN	291	296	GhknCL
	O95279 KCNK5_HUMAN	122	127	GvplCL		384	P09326 CD48_HUMAN	5	10	GwdsCL
314	O95294 RASL1_HUMAN	130	135	GqgrCL		385	P09341 GROA_HUMAN	81	86	GrkaCL
315	O95342 ABCBB_HUMAN	327	332	GfvwCL	35	386	P09848 LPH_HUMAN	1846	1851	GphaCL
	O95373 IPO7_HUMAN	147	152	GillCL			P10071 GLI3_HUMAN	1359	1364	GpesCL
	O95396 MOCS3_HUMAN	250	255	GvlgCL			P10109 ADX_HUMAN	151	156	GeqiCL
	O95405 ZFYV9_HUMAN	137	142	GnlaCL		389	P10145 IL8_HUMAN	73	78	GrelCL
	O95477 ABCA1_HUMAN O95500 CLD14_HUMAN	2120 178	2125 183	GrfrCL GtllCL			P10635 CP2D6_HUMAN P10646 TFPI1_HUMAN	439 213	444 218	GrraCL GpswCL
	O95551 TTRAP_HUMAN	217	222	GnelCL			P10720 PF4V_HUMAN	40	45	GdlqCL
	O95602 RPA1_HUMAN	1556	1561	GitrCL	40		P10720 PF4V_HUMAN	82	87	GrkiCL
	O95620 DUS4L_HUMAN	125	130	GygaCL			P10745 IRBP_HUMAN	328	333	GvvhCL
	O95633 FSTL3_HUMAN	88	93	GlvhCL				903	908	GqceCL
	O95671 ASML_HUMAN	588	593	GeyqCL			P11362 FGFR1_HUMAN	337	342	GeytCL
	O95714 HERC2_HUMAN	717	722	GsthCL			P11717 MPRI_HUMAN	231	236	GtaaCL
	O95714 HERC2_HUMAN	3265	3270	GalhCL	45		_	155	160	GlgdCL
	O95714 HERC2_HUMAN	4047	4052	GgkhCL	45		P13473 LAMP2_HUMAN	228	233	GndtCL
	O95715 SCYBE_HUMAN O95780 ZN682 HUMAN	68	73	GqehCL			P13498 CY24A_HUMAN P13569 CFTR_HUMAN	45	50	GvfvCL
	O95803 NDST3_HUMAN	132 815	137 820	GlnqCL GktkCL			P13686 PPA5_HUMAN	124 215	129 220	GiglCL GpthCL
	O95858 TSN15_HUMAN	285	290	GtgcCL				49	54	GevsCL
	O95873 CF047_HUMAN	171	176	GpeeCL			P13807 GYS1_HUMAN	185	190	GvglCL
	O95886 DLGP3_HUMAN	284	289	GgpfCL	50		P13861 KAP2_HUMAN	354	359	GdvkCL
335	O95967 FBLN4_HUMAN	76	81	GgylCL		406	P14222 PERF_HUMAN	530	535	GggtCL
336	O95977 EDG6_HUMAN	333	338	GpgdCL		407	P14543 NID1_HUMAN	24	29	GpvgCL
	O96006 ZBED1_HUMAN	221	226	GapnCL			P14867 GBRA1_HUMAN	6	11	GlsdCL
	O96008 TOM40_HUMAN	72	77	GacgCL		409	P15151 PVR_HUMAN	119	124	GnytCL
	O96009 NAPSA_HUMAN	350	355	GvrlCL		410	<del>-</del>	446	451	GmrqCL
340	P00505 AATM_HUMAN P00750 TPA_HUMAN	268 515	273 520	GinvCL GplvCL	55		P15692 VEGFA_HUMAN P16109 LYAM3_HUMAN	168 271	173 276	GarcCL GnmiCL
	P00751 CFAB_HUMAN	288	293	GakkCL			P16112 PGCA_HUMAN	2183	2188	GhviCL
	P01130 LDLR_HUMAN	314	319	GtneCL			P16581 LYAM2_HUMAN	376	381	Giviel
	P01133 EGF_HUMAN	741	746	GadpCL			P17038 ZNF43_HUMAN	127	132	GfnqCL
	P01266 THYG_HUMAN	2020	2025	GevtCL			P17040 ZNF31_HUMAN	184	189	GnsvCL
346	P01375 TNFA_HUMAN	26	31	GsrrCL			P17936 IBP3_HUMAN	66	71	GcgcCL
	P01730 CD4_HUMAN	366	371	GmwqCL	60		P18510 IL1RA_HUMAN	87	92	GgkmCL
	P01833 PIGR_HUMAN	437	442	GfywCL				674	679	GeneCL
	P02775 SCYB7_HUMAN	101	106	GrkiCL			P18577 RHCE_HUMAN	306	311	GgakCL
	P02776 PLF4_HUMAN	37	42	GdlqCL			P19099 C11B2_HUMAN	446	451	GmrqCL
	P02776 PLF4_HUMAN	79 70	84 75	GrkiCL GolgeCL			P19224 UD16_HUMAN	512	517	GyrkCL GdraCL
	P02778 SCYBA_HUMAN P02787 TRFE_HUMAN	70 209	75 214	GekrCL GafkCL	65		P19367 HXK1_HUMAN P19835 CEL_HUMAN	713 96	718 101	GdngCL GdedCL
	P02787 TRFE_HUMAN	538	543	GairCL GafrCL	00		P19875 MIP2A_HUMAN	96 81	86	GqeaCL GqkaCL
JJ+	102/0/ITRI D_HOWAIT	220	273	Janel		743	1150/5/WIII 221_IIOWAIN	01	00	Oqnacı

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11 GravCL

## TABLE 4-continued

## **68** TABLE 4-continued

	TABLE 4	-continued			_		TABLE 4	-continued		
	Motif: G	XCs -X(3)-C-L ocations: 1337	170		<b>-</b> 5		Motif: G-	Cs -X(3)-C-L ocations: 1337		
	Number of Diffe.	rent i fotems. i	170		-		Number of Differ	ent i iotems. i	.170	
#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence		#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
	P19876 MIP2B_HUMAN	81	86	GkkaCL	10		P35367 HRH1_HUMAN	96	101	GrplCL
	P19883 FST_HUMAN P20062 TCO2_HUMAN	252 79	257 84	GgkkCL GyqqCL		498 499	P35452 HXD12_HUMAN P35498 SCN1A_HUMAN	176 964	181 969	GvasCL GqamCL
	P20273 CD22_HUMAN	691	696	GlgsCL			P35499 SCN4A_HUMAN	774	779	GqamCL
	P20648 ATP4A_HUMAN	108	113	GglqCL			P35503 UD13_HUMAN	514	519	GyrkCL
	P20701 ITAL_HUMAN	76	81	GtghCL		502	_	514	519	GyrkCL
	P20701 ITAL_HUMAN P20813 CP2B6_HUMAN	1150 432	1155 437	GdpgCL GkriCL	15		P35555 FBN1_HUMAN P35555 FBN1_HUMAN	1259 1385	1264 1390	GeyrCL GsyrCL
	P20916 MAG_HUMAN	301	306	GvyaCL		505		1416	1421	GngqCL
435	P20929 NEBU_HUMAN	4517	4522	GvvhCL			P35555 FBN1_HUMAN	1870	1875	GsfyCL
	P21554 CNR1_HUMAN	427	432	GdsdCL			P35555 FBN1_HUMAN	2034	2039	GsfkCL
	P21580 TNAP3_HUMAN P21802 FGFR2_HUMAN	99 5	104 10	GdgnCL GrfiCL			P35556 FBN2_HUMAN P35556 FBN2_HUMAN	1303 1952	1308 1957	GeyrCL GsynCL
	P21802 FGFR2_HUMAN	338	343	GeytCL	20		P35556 FBN2_HUMAN	1994	1999	GsfkCL
	P21817 RYR1_HUMAN	840	845	GpsrCL			P35556 FBN2_HUMAN	2076	2081	GgfqCL
	P21860 ERBB3_HUMAN P21964 COMT_HUMAN	513 30	518 35	GpgqCL GwglCL			P35590 TIE1_HUMAN P35916 VGFR3_HUMAN	280 4	285 9	GltfCL GaalCL
	P22064 LTB1S_HUMAN	938	943	GsfrCL			P35968 VGFR2_HUMAN	638	643	GdyvCL
	P22064 LTB1S_HUMAN	1359	1364	GsykCL			P36509 UD12_HUMAN	510	515	GyrkCL
	P22105 TENX_HUMAN	565	570	GrgqCL	25		P36888 FLT3_HUMAN	99	104	GnisCL
	P22309 UD11_HUMAN P22309 UD11_HUMAN	276 513	281 518	GginCL GyrkCL			P37058 DHB3_HUMAN P38398 BRCA1_HUMAN	13 949	18 954	GllvCL GsrfCL
	P22310 UD14_HUMAN	514	519	GyrkCL			P38571 LICH_HUMAN	7	12	GlvvCL
	P22314 UBE1_HUMAN	230	235	GvvtCL			P38571 LICH_HUMAN	58	63	GyilCL
	P22455 FGFR4_HUMAN	97	102	GrylCL			P38606 VATA1_HUMAN	390	395	GrvkCL
	P22455 FGFR4_HUMAN P22455 FGFR4_HUMAN	220 329	225 334	GtytCL GeytCL	30		P38607 VATA2_HUMAN P39059 COFA1_HUMAN	388 8	393 13	GrvkCL GqcwCL
	P22607 FGFR3_HUMAN	335	340	GeytCL			P40205 NCYM_HUMAN	100	105	GrppCL
	P22680 CP7A1_HUMAN	330	335	GnpiCL		525	P40939 ECHA_HUMAN	709	714	GfppCL
	P22732 GTR5_HUMAN	348	353	GfsiCL			P41217 OX2G_HUMAN	117	122	GcymCL
	P23142 FBLN1_HUMAN P23142 FBLN1_HUMAN	269 547	274 552	GihnCL GgfrCL			P42331 RHG25_HUMAN P42345 FRAP_HUMAN	4 1479	9 1484	GqsaCL GrmrCL
	P23416 GLRA2_HUMAN	376	381	GmghCL	35		P42785 PCP_HUMAN	339	344	GqvkCL
	P23759 PAX7_HUMAN	466	471	GqseCL			P42830 SCYB5_HUMAN	87	92	GkeiCL
	P24386 RAE1_HUMAN	395 475	400 480	GgiyCL GargCI			P42892 ECE1_HUMAN	79 334	84 339	GlvaCL
	P24557 THAS_HUMAN P24592 IBP6_HUMAN	100	105	GprsCL GrgrCL		533	P43378 PTN9_HUMAN P43403 ZAP70_HUMAN	113	118	GdvpCL GvfdCL
	P24593 IBP5_HUMAN	96	101	GrgvCL	40		P43403 ZAP70_HUMAN	245	250	GliyCL
	P24821 TENA_HUMAN	143	148	GageCL	40		P46379 BAT3_HUMAN	872	877	GlfeCL
	P24903 CP2F1_HUMAN P25205 MCM3_HUMAN	432 239	437 244	GrrlCL GtyrCL			P46531 NOTC1_HUMAN P47775 GPR12_HUMAN	1354 166	1359 171	GslrCL GtsiCL
	P25874 UCP1_HUMAN	21	26	GiaaCL			P47804 RGR_HUMAN	275	280	GiwqCL
468	P25940 CO5A3_HUMAN	1581	1586	GgetCL				204	209	GgklCL
	P26374 RAE2_HUMAN	397	402	GgiyCL	45		P48052 CBPA2_HUMAN	12	17	GhiyCL
	P26951 IL3RA_HUMAN P27487 DPP4_HUMAN	363 335	368 340	GleeCL GrwnCL	43		P48059 PINC_HUMAN P48067 SC6A9_HUMAN	176 457	181 462	GelyCL GtqfCL
	P27540 ARNT_HUMAN	332	337	GskfCL			P48230 T4S4_HUMAN	5	10	GcarCL
473	P27987 IP3KB_HUMAN	284	289	GtrsCL			P48745 NOV_HUMAN	60	65	GcscCL
	P28332 ADH6_HUMAN	237 709	242 714	GateCL GklpCI			P49247 RPIA_HUMAN P49327 FAS_HUMAN	100 1455	105 1460	GgggCL GlvnCL
	P28340 DPOD1_HUMAN P29274 AA2AR_HUMAN	162	714 167	GklpCL GqvaCL	50		P49588 SYAC_HUMAN	1455 897	902	GkitCL
477	P29353 SHC1_HUMAN	570	575	GselCL	50	548	P49640 EVX1_HUMAN	345	350	GpcsCL
	P29459 IL12A_HUMAN	33	38	GmfpCL			P49641 MA2A2_HUMAN	862	867	GwrgCL
	P30040 ERP29_HUMAN P30530 UFO_HUMAN	153 106	158 111	GmpgCL GqyqCL			P49646 YYY1_HUMAN P49753 ACOT2_HUMAN	393 296	398 301	GetpCL GgelCL
	P30532 ACHA5_HUMAN	279	284	GekiCL			P49903 SPS1_HUMAN	323	328	GlliCL
482	P30566 PUR8_HUMAN	169	174	GkrcCL	55	553	P49910 ZN165_HUMAN	32	37	GqdtCL
	P31323 KAP3_HUMAN	368	373	GtvkCL			P50851 LRBA_HUMAN	2736	2741	GpenCL GodoCI
	P32004 L1CAM_HUMAN P32004 L1CAM_HUMAN	308 493	313 498	GeyrCL GryfCL			P51151 RAB9A_HUMAN P51168 SCNNB_HUMAN	79 532	84 537	GsdcCL GsvlCL
	P32314 FOXN2_HUMAN	319	324	GirtCL			P51589 CP2J2_HUMAN	444	449	GkraCL
	P32418 NAC1_HUMAN	414	419	GtyqCL			P51606 RENBP_HUMAN	37	42	GfftCL
	P32929 CGL_HUMAN P32970 TNFL7_HUMAN	80 29	85 34	GakyCL GlviCL	60		P51674 GPM6A_HUMAN P51685 CCR8_HUMAN	170 150	175 155	GanlCL GttlCL
	P33402 GCYA2_HUMAN	284	289	GreeCL			P51790 CLCN3_HUMAN	520	525	GaaaCL
491	P34913 HYES_HUMAN	258	263	GpavCL		562	P51790 CLCN3_HUMAN	723	728	GlrqCL
	P34981 TRFR_HUMAN	94	99	GyvgCL			P51793 CLCN4_HUMAN	520 721	525 726	GaaaCL
	P34998 CRFR1_HUMAN P35227 PCGF2_HUMAN	83 316	88 321	GyreCL GslnCL			P51793 CLCN4_HUMAN P51795 CLCN5_HUMAN	721 506	726 511	GlrqCL GaaaCL
	P35251 RFC1_HUMAN	402	407	GaenCL	65		P51795 CLCN5_HUMAN	707	712	GlrqCL
	P35270 SPRE_HUMAN	6	11	GravCL			P51800 CLCKA HUMAN	613	618	GhaaCL

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613

618 GhqqCL

TABLE 4-continued

	Motif: G	XCs -X(3)-C-L ocations: 1337 rent Proteins: 1	170		5				170	
#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence		#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
568	P51801 CLCKB_HUMAN	613	618	GhqqCL	10	639	Q01064 PDE1B_HUMAN	243	248	GmvhCL
569	P51957 NEK4_HUMAN	322	327	GegkCL		640	Q01433 AMPD2_HUMAN	103	108	GpapCL
	P52306 GDS1_HUMAN	25	30	GeldCL		641	_	107	112	GvyqCL
	P52306 GDS1_HUMAN P52429 DGKE HUMAN	265	270	GlveCL		642 643	Q02246 CNTN2_HUMAN	203	208	GnysCL
	P52744 ZN138_HUMAN	411 48	416 53	GtkdCL GlnqCL		644	Q02318 CP27A_HUMAN Q02985 FHR3_HUMAN	472 188	477 193	GvraCL GsitCL
	P52789 HXK2_HUMAN	713	718	GdngCL	1.5	645	Q03923 ZNF85_HUMAN	133	138	GlnqCL
	P52803 EFNA5_HUMAN	147	152	GrrsCL	15	646		184	189	GmisCL
	P52823 STC1_HUMAN	55	60	GafaCL		647	Q03924 ZN117_HUMAN	103	108	GlnqCL
	P52848INDST1_HUMAN	824	829	GktkCL		648	Q03936 ZNF92_HUMAN	132	137	GlnqCL
578 579	P52849 NDST2_HUMAN P52849 NDST2_HUMAN	302 823	307 828	GkrlCL GktrCL		649 650	Q03938 ZNF90_HUMAN Q04721 NOTC2_HUMAN	132 476	137 481	GlnqCL GgftCL
	P52961 NAR1_HUMAN	220	225	GiwtCL		651	Q05469 LIPS_HUMAN	716	721	GeriCL
	P53355 DAPK1_HUMAN	1326	1331	GkdwCL	20	652		530	535	GktfCL
	P54132 BLM_HUMAN	891	896	GiiyCL		653	Q06732 ZN11B_HUMAN	531	536	GktfCL
	P54277 PMS1_HUMAN	837	842	GmanCL		654	Q07325 SCYB9_HUMAN	70	75	GvqtCL
	P54750 PDE1A_HUMAN P54753 EPHB3 HUMAN	32 297	37 302	GilrCL GegpCL		655 656	Q07617 SPAG1_HUMAN Q07954 LRP1_HUMAN	133 875	138 880	GsnsCL GdndCL
	P54826 GAS1 HUMAN	19	24	GawlCL		657	Q07954 LRP1_HUMAN	3001	3006	GsykCL
	P55160 NCKPL_HUMAN	938	943	GpieCL	25	658	Q08629 TICN1_HUMAN	178	183	GpcpCL
588	P55268 LAMB2_HUMAN	501	506	GedrCL		659	Q09428 ABCC8_HUMAN	1073	1078	GivlCL
	P55268 LAMB2_HUMAN	1063	1068	GqcpCL		660	Q10471 GALT2_HUMAN	535	540	GsnlCL
	P56192 SYMC_HUMAN P56749 CLD12_HUMAN	8 63	13 68	GvpgCL GssdCL		661 662	_	63 66	68 71	GdgpCL GgylCL
	P57077 TAK1L_HUMAN	68	73	GflkCL		663	Q12809 KCNH2_HUMAN	719	724	GfpeCL
	P57679 EVC_HUMAN	683	688	GssqCL	30	664	*	48	53	GeptCL
	P58215 LOXL3_HUMAN	13	18	GIIICL		665	Q12852 M3K12_HUMAN	90	95	GlfgCL
	P58397 ATS12_HUMAN	447	452	GwgfCL		666	Q12860 CNTN1_HUMAN	110	115	GiyyCL
	P58418 USH3A_HUMAN P58512 CU067_HUMAN	69 166	74 171	GscgCL GfpaCL		667 668	_	988 387	993 392	GctlCL GykmCL
	P59047 NALP5_HUMAN	64	69	GlqwCL		669	Q12986 NFX1_HUMAN	537	542	GdfsCL
599	P59510 ATS20_HUMAN	458	463	GygeCL	35	670	Q13077 TRAF1_HUMAN	302	307	GyklCL
	P60370 KR105_HUMAN	32	37	GtapCL	-	671	_	48	53	GlrpCL
	P60371 KR106_HUMAN P60409 KR107_HUMAN	16 16	21 21	GsrvCL GsrvCL		672 673	Q13200 PSMD2_HUMAN Q13224 NMDE2_HUMAN	135 584	140 589	GereCL GynrCL
	P60413 KR10C_HUMAN	11	16	GsrvCL			Q13224 NMDE2_HUMAN	1392	1397	GddqCL
	P60602 CT052_HUMAN	38	43	GtfsCL		675	•	136	141	GinrCL
	P61011 SRP54_HUMAN	129	134	GwktCL	40	676	_	305	310	GghcCL
	P61550 ENT1_HUMAN P61619 S61A1 HUMAN	343 143	348 148	GnasCL	70	677 678	Q13308 PTK7_HUMAN Q13309 SKP2_HUMAN	429 107	434 112	GyldCL GifsCL
	_	46	51	GagiCL GesvCL		679	Q13322 GRB10_HUMAN	219	224	GlerCL
	P62312 LSM6_HUMAN	32	37	GvlaCL		680	_	253	258	GgagCL
	P62714 PP2AB_HUMAN	161	166	GqifCL		681	_	200	205	GemiCL
	P67775 PP2AA_HUMAN	161	166	GqifCL	45		_	594	599	GlfsCL
	P68371 TBB2C_HUMAN P69849 NOMO3_HUMAN	235 507	240 512	GvttCL GkvsCL	43		Q13410 BT1A1_HUMAN Q13444 ADA15_HUMAN	8 405	13 410	GlprCL GmgsCL
	P78310 CXAR_HUMAN	219	224	GsdqCL		685	-	105	110	GglkCL
	P78324 SHPS1_HUMAN	12	17	GpllCL		686	Q13485 SMAD4_HUMAN	359	364	GdrfCL
	P78325 ADAM8_HUMAN	101	106	GqdhCL		687	` =	472	477	GpppCL
	P78346 RPP30_HUMAN P78357 CNTP1_HUMAN	253 1205	258 1210	GdedCL GfsgCL	50	688 689	_	819 876	824 881	GgmpCL GgdiCL
	P78423 X3CL1_HUMAN	350	355	GlifCL	50	690	•	89	94	GevfCL
	P78504 JAG1_HUMAN	898	903	GprpCL		691	-	23	28	GhhcCL
	P78509 RELN_HUMAN	2862	2867	GhgdCL		692		300	305	GlphCL
	P78524 ST5_HUMAN	127 286	132	GvaaCL GaatCL		693 694	Q13698 CAC1S_HUMAN	1210 449	1215 454	GglyCL
	P78549 NTHL1_HUMAN P78559 MAP1A_HUMAN	2433	291 2438	GqqtCL GpqgCX			Q13751 LAMB3_HUMAN Q13772 NCOA4_HUMAN	97	102	GrelCL GqfnCL
	P80162 SCYB6_HUMAN	87	92	GkqvCL	55	696	*	364	369	GnlkCL
	P82279 CRUM1_HUMAN	1092	1097	GlqgCL		697		159	164	GrrdCL
	P83105 HTRA4_HUMAN	10	15	GlgrCL			Q13822 ENPP2_HUMAN	21	26	GvniCL
	P98088 MUC5A_HUMAN P98095 FBLN2_HUMAN	853 1047	858 1052	GcprCL GsfrCL		699 700	Q13885 TBB2A_HUMAN Q14008 CKAP5_HUMAN	235 109	240 114	GvttCL GieiCL
	P98153 IDD_HUMAN	289	294	GddpCL			Q14008 CKAP5_HUMAN	1237	1242	GvigCL
	P98160 PGBM_HUMAN	3181	3186	GtyvCL	60		Q14114 LRP8_HUMAN	175	180	GnrsCL
	P98161 PKD1_HUMAN	649	654	GaniCL			Q14114 LRP8_HUMAN	336	341	GlneCL
	P98164 LRP2_HUMAN P98164 LRP2_HUMAN	1252 3819	1257 3824	GhpdCL GsadCL		704 705	Q14159 K0146_HUMAN Q14264 ENR1_HUMAN	513 358	518 363	GtraCL GeltCL
	P98173 FAM3A_HUMAN	83	3824 88	GsadCL GpkiCL		706	*	1649	1654	GlgaCL
	P98194 AT2C1_HUMAN	158	163	GdtvCL			Q14344 GNA13_HUMAN	314	319	GdphCL
637	Q00872 MYPC1_HUMAN	447	452	GkeiCL	65	708	Q14392 LRC32_HUMAN	360	365	GslpCL
638	Q00973 B4GN1_HUMAN	408	413	GlgnCL		709	Q14393 GAS6_HUMAN	138	143	GnffCL

### TABLE 4-continued

CXCs
Motif: G-X(3)-C-L
Number of Locations: 1337
Number of Different Proteins: 1170
5

Number of Diffe	rent Proteins: I	.170				Number of Differen	nt Proteins: I	170	
	First	Last					First	Last	
Accession	Amino	Amino	~			Accession	Amino	Amino	~
# Number Protein Name	acid	acid	Sequence		#	Number Protein Name	acid	acid	Sequence
710 014202/5 456 11134431	217	222	GOI	<b>-</b> '	701	OSTIMOLODI DA O TITTO A A NI	242	240	G-f-GI
710 Q14393 GAS6_HUMAN	217	222	GsysCL	10	781	•	243	248	GsfrCL
711 Q14435 GALT3_HUMAN	93	98	GerpCL		782		507	512	GkvsCL
712 Q14435 GALT3_HUMAN	513	518	GqplCL		783	Q5JQC9 AKAP4_HUMAN	242	247	GkskCL
713 Q14451 GRB7_HUMAN	517	522	GilpCL		784	_	132	137	GlkqCL
714 Q14520 HABP2_HUMAN	121	126	GrgqCL		785	Q5JWF2 GNAS1_HUMAN	2	7	GvrnCL
715 Q14524 SCN5A_HUMAN	911	916	GqslCL		786	Q5JWF2 GNAS1_HUMAN	584	589	GtsgCL
716 Q14566 MCM6_HUMAN	154	159	GtflCL	15	787	Q5JWF8 CT134_HUMAN	111	116	GccvCL
717 Q14593 ZN273_HUMAN	100	105	GlngCL	13	788	Q5MJ68 SPDYC_HUMAN	138	143	GkdwCL
718 Q14656 ITBA1_HUMAN	197	202	GvlsCL		789	Q5NUL3 GP120_HUMAN	72	77	GataCL
719 Q14669 TRIPC_HUMAN	562	567	GladCL			Q5SRN2 CF010_HUMAN	117	122	GsikCL
720 Q14669 TRIPC_HUMAN	1136	1141	GgaeCL			Q5T2D3 OTUD3_HUMAN	72	77	GdgnCL
721 Q14703 MBTP1_HUMAN	845	850	GdsnCL			Q5T5C0 STXB5_HUMAN	322	327	GrrpCL
722 Q14714 SSPN_HUMAN	91	96	GiivCL		793		72	77	
. –				20		_			GggcCL
723 Q14766 LTB1L_HUMAN	1139	1144	GsfrCL			Q5T752 LCE1D_HUMAN	68	73	GggcCL
724 Q14766 LTB1L_HUMAN	1560	1565	GsykCL		795	_	72	77	GggcCL
725 Q14767 LTBP2_HUMAN	990	995	GsytCL			Q5T754 LCE1F_HUMAN	72	77	GggcCL
726 Q14767 LTBP2_HUMAN	1156	1161	GsyqCL			Q5T7P2 LCE1A_HUMAN	64	69	GggcCL
727 Q14767 LTBP2_HUMAN	1197	1202	GsffCL		798	Q5T7P3 LCE1B_HUMAN	72	77	GggcCL
728 Q14767 LTBP2_HUMAN	1238	1243	GsfnCL		799	Q5TA78 LCE4A_HUMAN	55	60	GggcCL
729 Q14767 LTBP2_HUMAN	1324	1329	GsfrCL	25	800	Q5TA79 LCE2A_HUMAN	64	69	GggcCL
730 Q14767 LTBP2_HUMAN	1366	1371	GsflCL		801	Q5TA82 LCE2D_HUMAN	68	73	GggcCL
731 Q14774 HLX1_HUMAN	483	488	GalgCL		802	Q5TCM9 LCE5A_HUMAN	64	69	GggcCL
732 Q14916 NPT1_HUMAN	110	115	GfalCL			Q5TEA3 CT194_HUMAN	465	470	GgngCL
733 Q14916 NPT1 HUMAN	207	212	GcavCL		804	` _	39	44	GnecCL
734 Q14940 SL9A5 HUMAN	576	581	GsgaCL			Q5THJ4 VP13D_HUMAN	1215	1220	GslgCL
735 Q14957 NMDE3_HUMAN	941	946	GpspCL	20		Q5VST9 OBSCN HUMAN	3315	3320	GdryCL
*				30		` -			
736 Q15021 CND1_HUMAN	730	735	GtiqCL		807		4189	4194	GvqwCL
737 Q15034 HERC3_HUMAN	145	150	GnwhCL		808	` -	5195	5200	GvyrCL
738 Q15048 LRC14_HUMAN	281	286	GrftCL			Q5VST9 OBSCN_HUMAN	6425	6430	GvytCL
739 Q15058 KIF14_HUMAN	438	443	GfntCL		810	Q5VT25 MRCKA_HUMAN	1325	1330	GaltCL
740 Q15061 WDR43_HUMAN	103	108	GtctCL		811	Q5VUA4 ZN318_HUMAN	1984	1989	GpspCL
741 Q15147 PLCB4_HUMAN	987	992	GgsnCL	35	812	Q5VZ18 SHE_HUMAN	8	13	GasaCL
742 Q15155 NOMO1_HUMAN	507	512	GkvsCL	33	813	Q5VZM2 RRAGB_HUMAN	366	371	GpkqCL
743 Q15274 NADC_HUMAN	92	97	GpahCL		814	Q5W111 CLLD6_HUMAN	50	55	GtggCL
744 Q15303 ERBB4_HUMAN	516	521	GpdqCL			Q5XUX1 FBXW9_HUMAN	184	189	GgslCL
745 Q15334 L2GL1_HUMAN	722	727	GvvrCL			Q5ZPR3 CD276_HUMAN	216	221	GtysCL
746 Q15399 TLR1_HUMAN	663	668	GmqiCL		817	•	434	439	GtysCL
747 Q15413 RYR3_HUMAN	229	234	GhdeCL			Q5ZPR3 CD276_HUMAN	472	477	GlsvCL
748 Q15413 RYR3_HUMAN	1656	1661	GlrtCL	40	819		216	221	GaehCL
	548	553			820	_	293	298	GkgrCL
_			GnpeCL			_			
750 Q15546 PAQRB_HUMAN	185	190	GliyCL		821	_	566	571	GfykCL
751 Q15633 TRBP2_HUMAN	321	326	GlcqCL		822	_	549	554	GnlfCL
752 Q15650 TRIP4_HUMAN	196	201	GsgpCL		823	_	190	195	GdgnCL
753 Q15652 JHD2C_HUMAN	1864	1869	GfvvCL		824		10	15	GlvlCL
754 Q15735 PI5PA_HUMAN	379	384	GpgrCL	45	825	Q6IS24 GLTL3_HUMAN	564	569	GtgrCL
755 Q15746 MYLK_HUMAN	229	234	GvytCL		826	Q6ISS4 LAIR2_HUMAN	10	15	GlvlCL
756 Q15746 MYLK_HUMAN	579	584	GtytCL		827	Q6ISS4 LAIR2_HUMAN	97	102	GlyrCL
757 Q15858 SCN9A_HUMAN	940	945	GqamCL		828	Q6N022 TEN4_HUMAN	139	144	GrssCL
758 Q15911 ATBF1_HUMAN	3527	3532	GsyhCL		829	Q6NUM9 RETST_HUMAN	366	371	GnarCL
759 Q16342 PDCD2_HUMAN	121	126	GesvCL			Q6P1M0 S27A4_HUMAN	297	302	GigqCL
760 O16363 LAMA4 HUMAN	1001	1006	GfvgCL	50	831		209	214	GniqCL
761 Q16549 PCSK7_HUMAN	16	21	GlptCL	50	832	•	96	101	GyalCL
762 Q16617 NKG7_HUMAN	15	20	GlmfCL		833		144	149	GqdfCL
_	437							305	
763 Q16647 PTGIS_HUMAN		442	GhnhCL		834		300		GvgqCL
764 Q16787 LAMA3_HUMAN	1526	1531	GvssCL		835		222	227	GgdaCL
765 Q30KQ9 DB111_HUMAN	60	65	GthcCL		836	-	397	402	GpvwCL
766 Q32MQ0 ZN750_HUMAN	121	126	GthrCL	55	837		794	799	GledCL
767 Q3KNT7 NSN5B_HUMAN	134	139	GaehCL	-	838	` _	22	27	GteiCL
768 Q3LI83 KR241_HUMAN	153	158	GqlnCL		839		498	503	GssgCL
769 Q3SYG4 PTHB1_HUMAN	822	827	GgrlCL		840	_	15	20	GgccCL
770 Q3T8J9 GON4L_HUMAN	1740	1745	GeadCL		841	Q6UWN5 LYPD5_HUMAN	15	20	GaalCL
771 Q495M9 USH1G_HUMAN	76	81	GhlhCL		842	Q6UX01 LMBRL_HUMAN	394	399	GnevCL
772 Q496M8 CI094 HUMAN	170	175	GefsCL		843	` -	199	204	GdgcCL
773 Q499Z4 ZN672_HUMAN	40	45	GrfrCL	60	844	` -	99	104	GilsCL
774 Q4G0F5 VP26B_HUMAN	167	172	GiedCL		845	_	127	132	GmwsCL
775 Q4KMG0 CDON_HUMAN	93	98	GyyqCL		846	•	226	231	GlyrCL
			GyyqCL		847				GarsCL
776 Q53G59 KLH12_HUMAN	426	431				_	455 517	460 522	
777 Q53H47 SETMR_HUMAN	72	77	GtcsCL		848	_	517	522	GnglCL
778 Q53R12 T4S20_HUMAN	213	218	GflgCL		849	Q6VVB1 NHLC1_HUMAN	47	52	GhvvCL
779 Q58EX2 SDK2_HUMAN	469	474	GtytCL	65	850	_	444	449	GrrhCL
780 Q5HYK3 COQ5_HUMAN	240	245	GrflCL		851	Q6W4X9 MUC6_HUMAN	1095	1100	GdceCL
•									

## TABLE 4-continued

CXCs Motif: G-X(3)-C-L Number of Locations: 1337 Number of Different Proteins: 1170 CXCs Motif: G-X(3)-C-L Number of Locations: 1337 Number of Different Proteins: 1170

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	Number of Differen	nt Proteins:	1170				Number of Differe	iii Fioteins. i	.170	
		First	Last					First	Last	
	Accession	Amino	Amino				Accession	Amino	Amino	
#	Number Protein Name	acid	acid	Sequence		#	Number   Protein Name	acid	acid	Sequence
852	Q6WN34 CRDL2_HUMAN	54	59	GlmyCL	10	923	Q8IWB7 WDFY1_HUMAN	200	205	GsvaCL
853	Q6ZN16 M3K15 HUMAN	82	87	GarqCL		924	O8IWN6 CX052 HUMAN	89	94	GskrCL
854	_	103	108	GgspCL			Q8IWV2 CNTN4_HUMAN	380	385	GmyqCL
							Q8IWY4 SCUB1 HUMAN			GsfqCL
855	_	141	146	GlstCL		926	_	342	347	1
	Q6ZRQ5 CF167_HUMAN	1116	1121	GilkCL		927	Q8IX30 SCUB3_HUMAN	337	342	GsfqCL
857	Q6ZSY5 PPR3F_HUMAN	647	652	GaevCL		928	Q8IXI1 MIRO2_HUMAN	515	520	GqtpCL
858	Q6ZV89 SH2D5_HUMAN	195	200	GghsCL	15	929	Q8IXW0 CK035_HUMAN	268	273	GslpCL
859	Q6ZVD8 PHLPL_HUMAN	5	10	GsrnCL	13	930	Q8IY26 PPAC2 HUMAN	149	154	GtlyCL
860	Q6ZW76 ANKS3_HUMAN	632	637	GgalCL		931	_	216	221	GvfyCL
861	Q75N90 FBN3_HUMAN	551	556	GsfsCL			Q8IYB9 ZN595_HUMAN	132	137	GvyqCL
	Q75N90 FBN3_HUMAN	1217	1222	GghrCL			Q8IYG6 LRC56_HUMAN	194	199	GnlvCL
863	Q75N90 FBN3_HUMAN	1826	1831	GsymCL		934	Q8IZ96 CKLF1_HUMAN	112	117	GgslCL
864	Q75N90 FBN3_HUMAN	1866	1871	GsynCL	20	935	Q8IZD0 SAM14_HUMAN	95	100	GgsfCL
865	Q75N90 FBN3_HUMAN	1908	1913	GsfhCL	20	936	Q8IZE3 PACE1_HUMAN	322	327	GetpCL
	Q75N90 FBN3_HUMAN	1990	1995	GsfqCL		937		521	526	GklÎCL
867	•	37	42	GewlCL			Q8IZJ1 UNC5B_HUMAN	547	552	GtfgCL
	_	230							322	
	Q7L0J3 SV2A_HUMAN		235	GrrqCL			Q8IZL8 PELP1_HUMAN	317		GlarCL
869	Q7L3T8 SYPM_HUMAN	149	154	GkeyCL		940	Q8IZY2 ABCA7_HUMAN	2001	2006	GrfrCL
870	Q7L622 K1333_HUMAN	310	315	GitdCL			Q8N122 RPTOR_HUMAN	549	554	GqeaCL
871	Q7LBC6 JHD2B_HUMAN	1049	1054	GfgvCL	25	942	Q8N122 RPTOR_HUMAN	1302	1307	GaisCL
872	Q7LBC6 JHD2B_HUMAN	1388	1393	GrllCL		943	Q8N1F7 NUP93_HUMAN	518	523	GdppCL
873	Q7RTN6 STRAD HUMAN	294	299	GtvpCL		944	Q8N1G0 ZN687_HUMAN	1133	1138	GaqqCL
	Q7RTP0 NIPA1_HUMAN	122	127	GklgCL			Q8N283 ANR35_HUMAN	65	70	GlteCL
	_									
875	Q7RTU9 STRC_HUMAN	1077	1082	GacsCL		946	Q8N283 ANR35_HUMAN	703	708	GlwdCL
	Q7RTX0 TS1R3_HUMAN	20	25	GaplCL		947	Q8N357 CB018_HUMAN	57	62	GefsCL
877	Q7Z2W7 TRPM8_HUMAN	652	657	GgsnCL	30	948	Q8N3C7 RSNL2_HUMAN	201	206	GavkCL
878	Q7Z333 SETX_HUMAN	1106	1111	GekkCL		949	Q8N3V7 SYNPO_HUMAN	28	33	GsyrCL
879	Q7Z3K3 POGZ HUMAN	749	754	GrqtCL		950	Q8N441 FGRL1_HUMAN	334	339	GmyiCL
	Q7Z3T1 OR2W3_HUMAN	108	113	GgveCL			Q8N442 GUF1 HUMAN	334	339	GdtlCL
881	Q7Z401 MYCPP_HUMAN	948	953	GsadCL			Q8N4B4 FBX39_HUMAN	114	119	GllsCL
							•			
	Q7Z460 CLAP1_HUMAN	146	151	GielCL		953	Q8N5D0 WDTC1_HUMAN	48	53	GevnCL
883	Q7Z4S6 KI21A_HUMAN	1493	1498	GpvmCL	35	954	_	9	14	GlgfCL
884	Q7Z5G4 GOGA7_HUMAN	68	73	GclaCL		955	Q8N655 CJ012_HUMAN	468	473	GdvkCL
885	Q7Z5K2 WAPL_HUMAN	850	855	GaerCL		956	Q8N6F8 WBS27_HUMAN	160	165	GglvCL
	Q7Z713 ANR37_HUMAN	75	80	GsleCL		957	Q8N6T3 ARFG1_HUMAN	38	43	GiwiCL
887	Q7Z7E8 UB2Q1_HUMAN	36	41	GpgpCL		958	Q8N6V9 TEX9_HUMAN	3	8	GrslCL
888		403	408			959	Q8N6Y1 PCD20_HUMAN	27	32	GpfsCL
	Q7Z7M1 GP144 HIDMAN			GcgwCL			•			•
	Q7Z7M1 GP144_HUMAN	343	348	GselCL	40		Q8N6Y1 PCD20_HUMAN	881	886	GiyiCL
890	Q86SG6 NEK8_HUMAN	418	423	GsngCL	70	961	Q8N726 CD2A2_HUMAN	160	165	GrarCL
891	Q86SQ6 GP123_HUMAN	1058	1063	GraaCL		962	Q8N813 CC056_HUMAN	42	47	GsctCL
892	Q86SQ6 GP123_HUMAN	1091	1096	GhasCL		963	Q8N895 ZN366_HUMAN	695	700	GrdeCL
893	Q86T20 CF001_HUMAN	75	80	GvldCL		964	Q8N8A2 ANR44_HUMAN	543	548	GhrqCL
894	Q86T65 DAAM2_HUMAN	570	575	GappCL		965	Q8N8A2 ANR44_HUMAN	645	650	GhtlCL
	•	234	239	GgelCL			•	112	117	
	Q86TX2 ACOT1_HUMAN				45		Q8N8Q9 NIPA2_HUMAN			GkigCL
896	Q86U44 MTA70_HUMAN	479	484	GkehCL	43		Q8N8R3 MCATL_HUMAN	133	138	GsldCL
897	Q86UE6 LRTM1_HUMAN	19	24	GvvlCL		968	Q8N9B4 ANR42_HUMAN	142	147	GrlgCL
898	Q86UK0 ABCAC_HUMAN	1251	1256	GwlcCL		969	Q8N9B4 ANR42_HUMAN	281	286	GhieCL
899	Q86UK5 LBN_HUMAN	26	31	GgrgCL		970	Q8N9L9 ACOT4_HUMAN	234	239	GadiCL
900	Q86UQ4 ABCAD_HUMAN	4056	4061	GppfCL		971	Q8NB46 ANR52_HUMAN	434	439	GnveCL
	Q86UQ4 ABCAD_HUMAN	4932	4937	GsfkCL			Q8NB46 ANR52_HUMAN	732	737	GcedCL
	Q86UU1 PHLB1_HUMAN	119	124	GemlCL	50		Q8NB46 ANR52_HUMAN	802	807	GhedCL
					50					
903	Q86UU1 PHLB1_HUMAN	1245	1250	GvdtCL		974	_	110	115	GyedCL
904		50	55	GnpnCL		975	Q8NBJ9 SIDT2_HUMAN	296	301	GmlfCL
905	Q86UW9 DTX2_HUMAN	347	352	GlpvCL		976	Q8NBV4 PPAC3_HUMAN	128	133	GtilCL
906	Q86V24 ADR2_HUMAN	190	195	GailCL		977	Q8NCL4 GALT6_HUMAN	505	510	GtnqCL
907	Q86V71 ZN429_HUMAN	132	137	GlnqCL		978	Q8NCL4 GALT6_HUMAN	593	598	GsgtCL
908	Q86VH4 LRTM4 HUMAN	271	276	GtfkCL		979	Q8NCN4 RN169 HUMAN	67	72	GeagCL
909	Q86WB7 UN93A_HUMAN	178	183	GasdCL	55	980	Q8NDX1 PSD4_HUMAN	183	188	GlkcCL
							_			
910	_	369	374	GyrsCL		981	Q8NDX1 PSD4_HUMAN	821	826	GedhCL
911	Q86WK7 AMGO3_HUMAN	348	353	GlfvCL		982	Q8NEN9 PDZD8_HUMAN	724	729	GgliCL
912	Q86WR7 CJ047_HUMAN	84	89	GgvcCL		983	Q8NFP4 MDGA1_HUMAN	622	627	GsaaCL
913	Q86X76 NIT1_HUMAN	288	293	GpglCL		984	Q8NFP9 NBEA_HUMAN	2819	2824	GpenCL
	Q86XN8 RKHD1_HUMAN	192	197	GtdvCL		985	Q8NFU7 CXXC6_HUMAN	1660	1665	GvtaCL
915	•	345	350	GlpvCL	60	986	Q8NG94 O11H1 HUMAN	112	117	GtseCL
	Q86Y56 HEAT2_HUMAN	271	276	GwllCL		987	Q8NG99 OR7G2_HUMAN	109	114	GlenCL
	•						•			
917	_	396	401	GlasCL		988	Q8NGC9 O11H4_HUMAN	118	123	GtteCL
918	_	503	508	GqpdCL		989	Q8NGH6 O52L2_HUMAN	96	101	GytvCL
919	Q8IUK8 CBLN2_HUMAN	27	32	GcgsCL		990	Q8NGH7 O52L1_HUMAN	96	101	GyivCL
920	Q8IUL8 CILP2_HUMAN	464	469	GcqkCL		991	Q8NGI2 O52N4_HUMAN	95	100	GfdeCL
921	Q8IVF6 ANR18_HUMAN	706	711	GykkCL	65	992	Q8NGJ0 OR5A1_HUMAN	111	116	GlseCL
	Q8IVH4 MMAA_HUMAN	96	101	GyraCL		993	Q8NGK5 O52M1_HUMAN	95	100	GldaCL
722	ZOLATITHATIATATTIONIATA	90	101	OqraCL		223	ZOLIOIZIONZIMII_HUMAN	23	100	OluaCL

# **76** TABLE 4-continued

CXCs
Motif: G-X(3)-C-L
Number of Locations: 1337
Number of Different Proteins: 1170

	Number of Differe	nt Proteins: 1	.170		- 3		Number of Differe	nt Proteins: 1	170	
#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence		#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
-004	OSNICROIORING HUMANI	112	117	CH-OI	-	1005	COSSOCIACO INDIANA	501	506	C10I
	Q8NGR9 OR1N2_HUMAN Q8NGS6 O13C3_HUMAN	112 108	117 113	GldnCL	10		Q92529 SHC3_HUMAN	581 248	586 253	GselCL
	Q8NGT2 O13J1_HUMAN	108	113	GsteCL GsteCL			Q92546 K0258_HUMAN Q92583 CCL17_HUMAN	30	35	GtvaCL GrecCL
	Q8NGT5 OR9A2_HUMAN	247	252	GygsCL			Q92621 NU205_HUMAN	950	955	GfveCL
	Q8NGT9 O2A42_HUMAN	107	112	GhseCL			Q92636 FAN_HUMAN	824	829	GtdgCL
	Q8NGU2 OR9A4_HUMAN	251	256	GygsCL			Q92673 SORL_HUMAN	1415	1420	GtageL
	Q8NGZ9 O2T10_HUMAN	109	114	GaecCL			Q92750 TAF4B_HUMAN	410	415	GaaiCL
	Q8NH09 OR8S1_HUMAN	109	114	GteaCL	15		Q92752 TENR_HUMAN	293	298	GgrgCL
	Q8NH19 O10AG_HUMAN	99	104	GgteCL			Q92782 DPF1_HUMAN	256	261	GhpsCL
1003	Q8NH40 OR6S1_HUMAN	66	71	GnlsCL		1074	Q92783 STAM1_HUMAN	41	46	GpkdCL
1004	Q8NHA8 OR1FC_HUMAN	50	55	GsdhCL		1075	Q92785 REQU_HUMAN	302	307	GhpsCL
	Q8NHU2 CT026_HUMAN	158	163	GnipCL			Q92794 MYST3_HUMAN	237	242	GhpsCL
	Q8NHU2 CT026_HUMAN	582	587	GfksCL	20		Q92832 NELL1_HUMAN	618	623	GgfdCL
	Q8NHW6 OTOSP_HUMAN	175	13	GlalCL			Q92854 SEM4D_HUMAN	620	625 375	GvyqCL
	Q8NHX4 SPTA3_HUMAN Q8NHY2 RFWD2_HUMAN	175 628	180 633	GsrsCL GkpyCL			Q92900 RENT1_HUMAN Q92932 PTPR2_HUMAN	370 35	373 40	GdeiCL GrlgCL
	Q8NHY3 GA2L2_HUMAN	463	468	GpaeCL			Q92932 PTPR2_HUMAN	634	639	GliyCL
	Q8TB24 RIN3_HUMAN	31	36	GmrlCL			Q92947 GCDH_HUMAN	285	290	GpfgCL
	Q8TB24 RIN3_HUMAN	971	976	GsppCL			Q92947 GCDH_HUMAN	346	351	GlhaCL
	Q8TCB7 METL6_HUMAN	89	94	GvgnCL	25		Q92952 KCNN1_HUMAN	361	366	GkgvCL
1014	Q8TCN5 ZN507_HUMAN	142	147	GmyrCL		1085	Q92956 TNR14_HUMAN	89	94	GlskCL
1015	Q8TCT7 PSL1_HUMAN	262	267	GlysCL		1086	Q92968 PEX13_HUMAN	216	221	GtvaCL
	Q8TCT7 PSL1_HUMAN	329	334	GiafCL			Q93038 TNR25_HUMAN	66	71	GnstCL
	Q8TCT8 PSL2_HUMAN	321	326	GiafCL			Q969L2 MAL2_HUMAN	37	42	GafvCL
	Q8TD26 CHD6_HUMAN	1627	1632	GnlcCL			Q969P0 IGSF8_HUMAN	402	407	GtyrCL
	Q8TD43 TRPM4_HUMAN	238	243	GthgCL	30		Q96A54 ADR1_HUMAN	179	184	GavlCL
	Q8TD43 TRPM4_HUMAN Q8TD43 TRPM4_HUMAN	306 650	311 655	GaadCL GdatCL			Q96AP0 ACD_HUMAN Q96AQ2 TM125 HUMAN	269 71	274 76	GalvCL GtvlCL
	Q8TD43 TRPM4_HUMAN	764	769	GgrrCL			Q96B26 EXOS8_HUMAN	230	235	GklcCL
	Q8TDJ6 DMXL2_HUMAN	188	193	GkddCL			Q96B86 RGMA_HUMAN	311	316	GlylCL
	Q8TDM6 DLG5_HUMAN	1672	1677	GvkdCL			Q96BD0 SO4A1_HUMAN	698	703	GletCL
	Q8TDN4 CABL1_HUMAN	135	140	GsgpCL	35		Q96CE8 T4S18_HUMAN	8	13	GclsCL
1026	Q8TDU6 GPBAR_HUMAN	81	86	GywsCL	33	1097	Q96CW5 GCP3_HUMAN	190	195	GvgdCL
	Q8TDU9 RL3R2_HUMAN	187	192	GvrlCL			Q96D59 RN183_HUMAN	95	100	GhqlCL
	Q8TDV0 GP151_HUMAN	183	188	GvemCL			Q96DN5 WDR67_HUMAN	52	57	GtgdCL
	Q8TDX9 PK1L1_HUMAN	317	322	GealCL			Q96DZ5 CLR59_HUMAN	212	217	GaakCL
	Q8TDY2 RBCC1_HUMAN	897	902	GelvCL			Q96EP1 CHFR_HUMAN	528	533	GcygCL
	Q8TDZ2 MICA1_HUMAN Q8TE49 OTU7A_HUMAN	743 206	748 211	GhfyCL GdgnCL	40		Q96EY5 F125A_HUMAN Q96EZ4 MYEOV_HUMAN	51 232	56 237	GyflCL GrraCL
	Q8TE58 ATS15_HUMAN	418	423	GhgdCL			Q96F46 I17RA_HUMAN	628	633	GraCL
	Q8TE85 GRHL3_HUMAN	429	434	GvkgCL			Q96GC6 ZN274_HUMAN	256	261	GttcCL
	Q8TEM1 PO210_HUMAN	1489	1494	GdvlCL			Q96H40 ZN486_HUMAN	132	137	GlnqCL
	Q8TF62 AT8B4_HUMAN	282	287	GfliCL			Q96H96 COQ2_HUMAN	172	177	GvllCL
1037	Q8TF76 HASP_HUMAN	190	195	GtsaCL		1108	Q96I82 KAZD1_HUMAN	249	254	GtyrCL
	Q8WTV0 SCRB1_HUMAN	319	324	GfcpCL	45		Q96IV0 NGLY1_HUMAN	70	75	GaveCL
	Q8WUB8 PHF10_HUMAN	320	325	GhpsCL			Q96IW7 SC22A_HUMAN	234	239	GtaaCL
	Q8WUM0 NU133_HUMAN	112	117	GgwaCL			Q96J02 ITCH_HUMAN	160	165	GvslCL
	Q8WWQ8 STAB2_HUMAN	1358	1363	GngiCL			Q96J94 PIWL1_HUMAN	674	679	GlkvCL
	Q8WWQ8 STAB2_HUMAN Q8WWX0 ASB5_HUMAN	2026 179	2031 184	GsgqCL GhheCL			Q96JH7 VCIP1_HUMAN Q96JK2 WDR22_HUMAN	215 178	220 183	GdghCL GepfCL
	Q8WWZ1 IL1FA_HUMAN	63	68	GgsrCL	50		Q96JT2 S45A3_HUMAN	27	32	GlevCL
	Q8WXI2 CNKR2_HUMAN	22	27	GlddCL	30		Q96JT2 S45A3_HUMAN	485	490	GrgiCL
	Q8WXI7 MUC16_HUMAN	22110	22115	GlitCL			Q96K31 CH076_HUMAN	98	103	GqarCL
	Q8WXK4 ASB12_HUMAN	75	80	GhlsCL			Q96KC8 DNJC1_HUMAN	228	233	GiwfCL
1048	Q8WXS8 ATS14_HUMAN	489	494	GyqtCL		1119	Q96KM6 K1196_HUMAN	782	787	GkyrCL
	Q8WXS8 ATS14_HUMAN	587	592	GgrpCL			Q96LC7 SIG10_HUMAN	373	378	GqslCL
	Q8WYB5 MYST4_HUMAN	244	249	GhpsCL	55		Q96LD4 TRI47_HUMAN	25	30	GhnfCL
	Q8WYP5 AHTF1_HUMAN	112	117	GsvlCL			Q96LQ0 CN050_HUMAN	366	371	GeprCL
	Q8WYP5 AHTF1_HUMAN	318	323	GnrkCL			Q96ME1 FXL18_HUMAN	352	357	GevhCL
	Q8WYP5 AHTF1_HUMAN Q8WZ42 TITIN HUMAN	526 4919	531 4924	GynrCL GkytCL			Q96ME7 ZN512_HUMAN Q96ME7 ZN512_HUMAN	320 438	325 443	GqpeCL GkykCL
	Q8WZ42 TITIN_HUMAN	5147	5152	GsavCL			Q96MU7 YTDC1_HUMAN	436 485	490	GtqlCL
	Q8WZ42 TITIN_HUMAN	7829	7834	GdysCL			Q96MU8 KREM1_HUMAN	53	58	GgkpCL
	Q8WZ42 TITIN_HUMAN	16742	16747	GagdCL	60		Q96NL3 ZN599_HUMAN	373	378	GktfCL
	Q8WZ42 TITIN_HUMAN	20237	20242	GtnvCL			Q96NX9 DACH2_HUMAN	585	590	GnyyCL
	Q8WZ73 RFFL_HUMAN	81	86	GprlCL			Q96P11 NSUN5_HUMAN	400	405	GaehCL
	Q8WZ74 CTTB2_HUMAN	924	929	GfknCL			Q96PH1 NOX5_HUMAN	272	277	GcgqCL
	Q92481 AP2B_HUMAN	379	384	GiqsCL			Q96PL5 ERMAP_HUMAN	122	127	GsyrCL
	Q92496 FHR4_HUMAN	130	135	GsitCL	C =		Q96PP9 GBP4_HUMAN	321	326	GavpCL
	Q92520 FAM3C_HUMAN	82	87	GpkiCL	65		Q96Q04 LMTK3_HUMAN	676	681	GacsCL
1064	Q92527 ANKR7_HUMAN	148	153	GeppCL		1135	Q96Q15 SMG1_HUMAN	2809	2814	GnvtCL

### TABLE 4-continued

### CXCs Motif: G-X(3)-C-L Number of Locations: 1337 Number of Different Proteins: 1170

	Number of Differe	nt Proteins: 1	.170		- 3		Number of Differe	ent Proteins: 1	170	
Access	ion r Protein Name	First Amino acid	Last Amino acid	Sequence		#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
1136 00600	27 ASB2_HUMAN	101	106	GavaCI	- 10	1207	Q9BYB4 GNB1L_HUMAN	163	1.69	GmnmCI
	27 ASB2_HUMAN	135	140	GqvgCL GhldCL	10		O9BYE0 HES7 HUMAN	95	168 100	GmpmCL GfreCL
	01 B3A4_HUMAN	455	460	GaafCL			Q9BYJ1 LOXE3_HUMAN	309	314	GqdtCL
	37 MTMR9 HUMAN	85	90	GmeeCL			O9BYK8 PR285 HUMAN	1908	1913	GfslCL
, ,	S1 TSN32_HUMAN	258	263	GpthCL			Q9BYT1 CT059_HUMAN	398	403	GswtCL
	J8 XPO6_HUMAN	413	418	GyfsCL			Q9BYX4 IFIH1_HUMAN	265	270	GsvsCL
1142 Q96R3	0 OR2V2_HUMAN	103	108	GlfvCL	15	1213	Q9BZ11 ADA33_HUMAN	400	405	GggaCL
•	/3 PCX1_HUMAN	696	701	GtvaCL	10		Q9BZ76 CNTP3_HUMAN	509	514	GfqgCL
•	V7 HMCN1_HUMAN	677	682	GiygCL			Q9BZ76 CNTP3_HUMAN	1163	1168	GftgCL
	V7 HMCN1_HUMAN	2546	2551	GrytCL			Q9BZC7 ABCA2_HUMAN	2262	2267	GrlrCL
	V7 HMCN1_HUMAN 43 CPXM1_HUMAN	3595 262	3600 267	GrytCL GgapCL			Q9BZF3 OSBL6_HUMAN Q9BZF9 UACA_HUMAN	554 79	559 84	GrraCL GnleCL
•	9 CP2S1_HUMAN	436	441	GkrvCL			Q9BZF9 UACA_HUMAN	112	117	GhalCL
	J4 OSBL9_HUMAN	542	547	GevsCL	20		Q9BZH6 BRWD2_HUMAN	79	84	GspyCL
	0 SCN2A_HUMAN	955	960	GqtmCL			Q9BZS1 FOXP3_HUMAN	228	233	GraqCL
,	6 NOTC4_HUMAN	216	221	GsfqCL			Q9BZY9 TRI31_HUMAN	32	37	GhnfCL
	6 NOTC4_HUMAN	375	380	GsfsCL			Q9BZZ2 SN_HUMAN	1507	1512	GmyhCL
1153 Q9946	6 NOTC4_HUMAN	414	419	GstlCL			Q9C004 SPY4_HUMAN	197	202	GtcmCL
,	6 NOTC4_HUMAN	457	462	GsfnCL			Q9C0A0 CNTP4_HUMAN	1163	1168	GftgCL
	6 NOTC4_HUMAN	609	614	GaffCL	25		Q9C0C6 K1737_HUMAN	47	52	GsseCL
•	6 NOTC4_HUMAN	787	792	GtfsCL			Q9GZK3 OR2B2_HUMAN	108	113	GsteCL
•	6 NOTC4_HUMAN 6 NOTC4_HUMAN	1121 1872	1126 1877	GgpdCL GggaCL			Q9GZR3 CFC1_HUMAN Q9GZY1 PBOV1_HUMAN	144 118	149 123	GalhCL GlecCL
,	8 M3K14_HUMAN	536	541	GhavCL			Q9H013 ADA19_HUMAN	400	405	GggmCL
	1 SPS2 HUMAN	373	378	GlliCL			Q9H093 NUAK2 HUMAN	587	592	GpgsCL
•	8 GPR20_HUMAN	115	120	GargCL	30		Q9H0A0 NAT10_HUMAN	654	659	GrfpCL
•	1 CDC6_HUMAN	207	212	GktaCL	50		Q9H0B3 K1683_HUMAN	578	583	GkirCL
1163 Q9975	8 ABCA3_HUMAN	1590	1595	GqfkCL		1234	Q9H0J9 PAR12_HUMAN	272	277	GdqiCL
	7 PMIP_HUMAN	277	282	GqlkCL			Q9H0M4 ZCPW1_HUMAN	249	254	GfgqCL
•	8 EBP2_HUMAN	52	57	GlkqCL			Q9H172 ABCG4_HUMAN	588	593	GdltCL
•	7 TBB4Q_HUMAN	235	240	GvttCL			Q9H195 MUC3B_HUMAN	545	550	GqcaCL
•	4 SC6A7_HUMAN	543	548 1469	GllsCL	35		Q9H1B7 CN004_HUMAN Q9H1D0 TRPV6_HUMAN	294 10	299 15	GgpaCL GlilCL
•	3 TEP1_HUMAN 3 TEP1_HUMAN	1464 1486	1491	GpfaCL GarlCL			Q9H1K4 GHC2_HUMAN	47	52	GmidCL
•	3 TEP1_HUMAN	1720	1725	GisaCL			Q9H1M3 DB129_HUMAN	23	28	GlmCL
	3 TEP1_HUMAN	2595	2600	GsvsCL			Q9H1M4 DB127_HUMAN	50	55	GrycCL
•	6 AKAP9_HUMAN	3063	3068	GllnCL			Q9H1P6 CT085_HUMAN	107	112	GlnkCL
1173 Q9BQ0	08 RSNB_HUMAN	2	7	GpssCL	40	1244	Q9H1R3 MYLK2_HUMAN	240	245	GqalCL
	G2 NUD12_HUMAN	348	353	GmftCL	40		Q9H1V8 S6A17_HUMAN	421	426	GldpCL
	R3 PRS27_HUMAN	231	236	GplvCL			Q9H221 ABCG8_HUMAN	421	426	GaeaCL
	S2 SYT15_HUMAN	23 373	28 378	GascCL GlsaCL			Q9H228 EDG8_HUMAN	347 571	352 576	GlrrCL
-	B3 PIGQ_HUMAN P4 WDR71_HUMAN	206	211	GrsaCL			Q9H252 KCNH6_HUMAN Q9H2D1 MFTC_HUMAN	64	69	GfpeCL GilhCL
•	Z2 TRI56_HUMAN	343	348	GpapCL			Q9H2G2 SLK_HUMAN	1208	1213	GeseCL
	36 ZPBP1_HUMAN	346	351	GaktCL	45		Q9H2M9 RBGPR_HUMAN	387	392	GesiCL
	40 SKIP_HUMAN	131	136	GvniCL			Q9H2S1 KCNN2_HUMAN	371	376	GkgvCL
1182 Q9BT	51 CU122_HUMAN	6	11	GfshCL		1253	Q9H2X9 S12A5_HUMAN	602	607	GmslCL
	F0 THUM2_HUMAN	407	412	GikkCL			Q9H2Y7 ZF106_HUMAN	975	980	GegnCL
	K1 NDC1_HUMAN	310	315	GsdeCL			Q9H324 ATS10_HUMAN	422	427	GlglCL
	Y5 ZN426_HUMAN	14	19	GdpvCL GwrgCI	50		Q9H324 ATS10_HUMAN	556 557	561	GgkyCL GggCI
	Y5 ZN426_HUMAN 38 WDR18_HUMAN	430 81	435 86	GypsCL GpvtCL	50		Q9H3D4 P73L_HUMAN Q9H3R1 NDST4_HUMAN	557 814	562 819	GcssCL GktkCL
	38 WDR18_HUMAN	139	144	GgkdCL			Q9H4F1 SIA7D HUMAN	29	34	GlplCL
	73 CP250_HUMAN	806	811	GevrCL			Q9H5U8 CX045_HUMAN	403	408	GfdsCL
	99 LRC61_HUMAN	113	118	GqlqCL			Q9H5V8 CDCP1_HUMAN	373	378	GcfvCL
	A1 TBB2B_HUMAN	235	240	GvttCL		1262	Q9H6E5 TUT1_HUMAN	15	20	GfrcCL
	H7 SIA7E_HUMAN	8	13	GlavCL	55		Q9H6R4 NOL6_HUMAN	391	396	GislCL
	K2 ALG8_HUMAN	361	366	GflrCL			Q9H792 SG269_HUMAN	1661	1666	GilqCL
	T7 CAR10_HUMAN	916	921	GkkhCL			Q9H7F0 AT133_HUMAN	109	114	GhavCL
	U0 NADAP_HUMAN U0 NADAP_HUMAN	185 196	190 201	GtsyCL GcdvCL			Q9H7M9 GI24_HUMAN Q9H808 TLE6_HUMAN	142 315	147 320	GlycCL GpdaCL
	V1 BOC_HUMAN	1053	1058	Genvel			Q9H8X2 IPPK HUMAN	110	115	GyamCL
	C9 BBS2_HUMAN	26	31	GthpCL			Q9H9S3 S61A2_HUMAN	143	148	GagiCL
•	L6 CAR14_HUMAN	850	855	GfkkCL	60		Q9HAF5 CO028_HUMAN	120	125	GvrmCL
	M7 PINK1_HUMAN	408	413	GgngCL			Q9HAS0 NJMU_HUMAN	123	128	GcyyCL
	R0 TGT_HUMAN	50	55	GcriCL			Q9HAT1 LMA1L_HUMAN	8	13	GplfCL
	S4 TMM59_HUMAN	229	234	GflrCL			Q9HAV4 XPO5_HUMAN	266	271	GaaeCL
•	ISTEX15_HUMAN	1099	1104	GekkCL			Q9HAW7 UD17_HUMAN	510	515	GyrkCL
-	U8 FHL17_HUMAN	78 562	83 567	GghiCL Getw.CL	65		Q9HAW8 UD110_HUMAN	510	515 515	GyrkCL GyrlcCL
•	15 EMR3_HUMAN 41 HDAC8_HUMAN	562 283	567 288	GctwCL GigkCL	0.5		Q9HAW9 UD18_HUMAN Q9HBX8 LGR6_HUMAN	510 550	515 555	GyrkCL GvlgCL
1200 Q9BY	TITUACO_HUMAN	283	288	OIRKCL		12//	CAMPAGE ON THE CAMPAIN	330	233	OVISCT

# **80** TABLE 4-continued

CXCs	
Motif: G-X(3)-C-L	
Number of Locations: 1337	
Number of Different Proteins: 1170	

Number of Differe		.170		_ 5	Number of Differe		170	
Accession # Number Protein Name	First Amino acid	Last Amino acid	Sequence		Accession # Number Protein Name	First Amino acid	Last Amino acid	Sequence
				-				
1278 Q9HBZ2 ARNT2_HUMAN 1279 Q9HC07 TM165_HUMAN	295 138	300 143	GskyCL GlmtCL	10	1349 Q9NYJ7 DLL3_HUMAN 1350 Q9NYQ6 CELR1_HUMAN	235 168	240 173	GecrCL GrpiCL
1280 Q9HC84 MUC5B_HUMAN	780	785	GklsCL		1351 Q9NYQ7 CELR3_HUMAN	2070	2075	GsdsCL
1281 Q9HC84 MUC5B_HUMAN	1281	1286	GlgaCL		1352 Q9NYQ8 FAT2_HUMAN	3908	3913	GfegCL
1282 Q9HCC6 HES4_HUMAN	113	118	GfheCL		1353 Q9NYQ8 FAT2_HUMAN	4285	4290	GggpCL
1283 Q9HCC9 ZFY28_HUMAN	555	560	GatnCL		1354 Q9NYW6 TA2R3_HUMAN	104	109	GvlyCL
1284 Q9HCE9 TM16H_HUMAN	541	546	GgrrCL	15	1355 Q9NZ56 FMN2_HUMAN	1694	1699	GkeqCL
1285 Q9HCM2 PLXA4_HUMAN 1286 Q9HCM4 E41L5_HUMAN	990 111	995 116	GkqpCL GspyCL		1356 Q9NZ71 RTEL1_HUMAN 1357 Q9NZ94 NLGN3_HUMAN	47 19	52 24	GktlCL GrslCL
1287 Q9HCU4 CELR2_HUMAN	1308	1313	GgytCL		1358 Q9NZH0 GPC5B_HUMAN	164	169	GlalCL
1288 Q9HCU4 CELR2_HUMAN	1757	1762	GfrgCL		1359 Q9NZH7 IL1F8_HUMAN	68	73	GkdlCL
1289 Q9HCU4 CELR2_HUMAN	1917	1922	GsptCL		1360 Q9NZL3 ZN224_HUMAN	550	555	GwasCL
1290 Q9NNW5 WDR6_HUMAN	460	465	GvvaCL	20	1361 Q9NZR2 LRP1B_HUMAN	866	871	GdddCL
1291 Q9NP73 GT281_HUMAN	82	87	GagsCL	20	1362 Q9NZR2 LRP1B_HUMAN	2987	2992	GtykCL
1292 Q9NP90 RAB9B_HUMAN 1293 Q9NPA1 KCMB3_HUMAN	79 121	84 126	GadcCL GkypCL		1363 Q9NZV5 SEPN1_HUMAN 1364 Q9P0K1 ADA22_HUMAN	273 429	278 434	GavaCL GggaCL
1294 Q9NPA3 M1IP1_HUMAN	58	63	GsggCL		1365 Q9P0K7 RAI14_HUMAN	64	69	GhveCL
1295 Q9NPD7 NRN1_HUMAN	37	42	GfsdCL		1366 Q9P0L1 ZN167_HUMAN	617	622	GlskCL
1296 Q9NPF8 CENA2_HUMAN	41	46	GifiCL		1367 Q9P0M9 RM27_HUMAN	84	89	GknkCL
1297 Q9NPG4 PCD12_HUMAN	807	812	GwdpCL	25	1368 Q9P0U3 SENP1_HUMAN	531	536	GvhwCL
1298 Q9NPH5 NOX4_HUMAN	51	56	GlglCL		1369 Q9P0X4 CAC1I_HUMAN	290	295	GrecCL
1299 Q9NQ25 SLAF7_HUMAN	3 125	130	GsptCL		1370 Q9P203 BTBD7_HUMAN 1371 Q9P255 ZN492_HUMAN	265	270	GnqnCL GlacCl
1300 Q9NQ30 ESM1_HUMAN 1301 Q9NQ75 CT032_HUMAN	50	130 55	GtgkCL GwwkCL		1371 Q9P233 ZN492_HUMAN 1372 Q9P273 TEN3_HUMAN	143 142	148 147	GlnqCL GrssCL
1302 Q9NQB0 TF7L2 HUMAN	492	497	GegsCL		1373 Q9P273 TEN3_HUMAN	1590	1595	GissCL
1303 Q9NQQ7 S35C2_HUMAN	302	307	GfalCL	30	1374 Q9P275 UBP36_HUMAN	824	829	GsetCL
1304 Q9NQS5 GPR84_HUMAN	195	200	GifyCL		1375 Q9P283 SEM5B_HUMAN	589	594	GgldCL
1305 Q9NQU5 PAK6_HUMAN	662	667	GlpeCL		1376 Q9P283 SEM5B_HUMAN	887	892	GediCL
1306 Q9NR09 BIRC6_HUMAN	511	516	GanpCL		1377 Q9P298 HIG1B_HUMAN	34	39	GlggCL
1307 Q9NR61 DLL4_HUMAN 1308 Q9NR63 CP26B_HUMAN	204 437	209 442	GnlsCL GvrtCL		1378 Q9P2B2 FPRP_HUMAN 1379 Q9P2C4 TM181_HUMAN	844 406	849 411	GllsCL GerkCL
1309 Q9NR81 ARHG3_HUMAN	203	208	GwlpCL		1380 Q9P2E3 ZNFX1_HUMAN	1162	1167	GelkCL
1310 Q9NR99 MXRA5_HUMAN	2414	2419	GnytCL	35	1381 Q9P2I0 CPSF2_HUMAN	759	764	GlegCL
1311 Q9NRI5 DISC1_HUMAN	23	28	GsrdCL		1382 Q9P2J9 PDP2_HUMAN	125	130	GvasCL
1312 Q9NRX5 SERC1_HUMAN	19	24	GsapCL		1383 Q9P2J9 PDP2_HUMAN	298	303	GmwsCL
1313 Q9NS15 LTBP3_HUMAN	846	851	GsyrCL		1384 Q9P2N4 ATS9_HUMAN	490	495	GygeCL
1314 Q9NS40 KCNH7_HUMAN 1315 Q9NS62 THSD1_HUMAN	722 419	727 424	GfpeCL GislCL		1385 Q9P2P6 STAR9_HUMAN 1386 Q9P2R3 ANFY1_HUMAN	715 720	720 725	GeadCL GpggCL
1316 Q9NSD7 RL3R1_HUMAN	243	248	GeelCL	40	1387 Q9P2R7 SUCB1_HUMAN	316	321	GpggCL GnigCL
1317 Q9NSI6 BRWD1_HUMAN	204	209	GsddCL		1388 Q9P2S2 NRX2A_HUMAN	1061	1066	GfqgCL
1318 Q9NSN8 SNTG1_HUMAN	242	247	GiiqCL		1389 Q9UBD9 CLCF1_HUMAN	10	15	GmlaCL
1319 Q9NST1 ADPN_HUMAN	24	29	GatrCL		1390 Q9UBE0 ULE1A_HUMAN	338	343	GiveCL
1320 Q9NST1 ADPN_HUMAN	97	102	GlekCL		1391 Q9UBG0 MRC2_HUMAN	50	55	GlqgCL
1321 Q9NT68 TEN2_HUMAN 1322 Q9NU22 MDN1_HUMAN	858 427	863 432	GlvdCL GrgdCL	45	1392 Q9UBG0 MRC2_HUMAN 1393 Q9UBG0 MRC2_HUMAN	89 938	94 943	GtmqCL GdqrCL
1323 Q9NUB4 CT141_HUMAN	156	161	GlafCL	10	1394 Q9UBG7 RBPSL_HUMAN	56	61	GuqreL
1324 Q9NUP1 CNO_HUMAN	67	72	GyaaCL		1395 Q9UBG7 RBPSL_HUMAN	326	331	GtylCL
1325 Q9NVE7 PANK4_HUMAN	304	309	GqlaCL		1396 Q9UBH0 IL1F5_HUMAN	63	68	GgsqCL
1326 Q9NVG8 TBC13_HUMAN	38	43	GglrCL		1397 Q9UBM4 OPT_HUMAN	124	129	GlptCL
1327 Q9NVX2 NLE1_HUMAN	474	479	GkdkCL		1398 Q9UBP5 HEY2_HUMAN	125	130	GfreCL
1328 Q9NW08 RPC2_HUMAN 1329 Q9NWT1 PK1IP_HUMAN	765 83	770 88	GfgrCL GtitCL	50	1399 Q9UBS8 RNF14_HUMAN 1400 Q9UBY5 EDG7_HUMAN	258 37	263 42	GqvqCL GtffCL
1330 Q9NWU5 RM22 HUMAN	142	147	GrgqCL		1401 Q9UBY8 CLN8_HUMAN	145	150	GflgCL
1331 Q9NWZ3 IRAK4_HUMAN	255	260	GddlCL		1402 Q9UDX3 S14L4_HUMAN	250	255	GnpkCL
1332 Q9NX02 NALP2_HUMAN	139	144	GnviCL		1403 Q9UDX3 S14L4_HUMAN	351	356	GsltCL
1333 Q9NXJ0 M4A12_HUMAN	106	111	GivlCL		1404 Q9UDX4 S14L3_HUMAN	250	255	GnpkCL
1334 Q9NXR5 ANR10_HUMAN	69	74	GkleCL	55	1405 Q9UGF7 O12D3_HUMAN	62 525	67	GnlsCL
1335 Q9NXR5 ANR10_HUMAN 1336 Q9NXS3 BTBD5 HUMAN	103 293	108 298	GhpqCL GlfaCL		1406 Q9UGI6 KCNN3_HUMAN 1407 Q9UGU5 HM2L1_HUMAN	525 567	530 572	GkgvCL GplaCL
1337 Q9NXW9 ALKB4_HUMAN	19	24	GirtCL		1408 Q9UHA7 IL1F6 HUMAN	69	74	GlnlCL
1338 Q9NY15 STAB1_HUMAN	122	127	GhgtCL		1409 Q9UHC6 CNTP2_HUMAN	1174	1179	GftgCL
1339 Q9NY15 STAB1_HUMAN	177	182	GdgsCL		1410 Q9UHD0 IL19_HUMAN	24	29	GlrrCL
1340 Q9NY15 STAB1_HUMAN	752	757	GngaCL	60	1411 Q9UHI8 ATS1_HUMAN	458	463	GhgeCL
1341 Q9NY15 STAB1_HUMAN	1256	1261	GssrCL	00	1412 Q9UHW9 S12A6_HUMAN	687	692	GmsiCL
1342 Q9NY15 STAB1_HUMAN 1343 Q9NY15 STAB1_HUMAN	1991 2250	1996 2255	GsgqCL GfhlCL		1413 Q9UHX3 EMR2_HUMAN 1414 Q9UIA9 XPO7_HUMAN	742 933	747 938	GctwCL GccsCL
1344 Q9NY33 DPP3_HUMAN	515	520	GlylCL		1415 Q9UIE0 ZN230_HUMAN	286	291	GksfCL
1345 Q9NY35 CLDND_HUMAN	213	218	GwsfCL		1416 Q9UIF8 BAZ2B_HUMAN	627	632	GmqwCL
1346 Q9NY46 SCN3A_HUMAN	956	961	GqtmCL		1417 Q9UIF9 BAZ2A_HUMAN	1006	1011	GpeeCL
1347 Q9NY91 SC5A4_HUMAN	507	512	GtgsCL	65	1418 Q9UIH9 KLF15_HUMAN	117	122	GehfCL
1348 Q9NY99 SNTG2_HUMAN	14	19	GrqgCL		1419 Q9UIR0 BTNL2_HUMAN	337	342	GqyrCL

81

TABLE 4-continued

82 TABLE 4-continued

CXCs		CXCs
Motif: G-X(3)-C-L		Motif: G-X(3)-C-L
Number of Locations: 1337		Number of Locations: 1337
Number of Different Proteins: 1170	5	Number of Different Proteins: 1170

Number of Different Proteins: 1170								
		Timet	Toot					
	Accession	First Amino	Last Amino					
#	Number Protein Name	acid	acid	Sequence				
		44.4		o equence				
	Q9UK10 ZN225_HUMAN	466	471	GwasCL				
1421	Q9UK11 ZN223_HUMAN	294	299	GksfCL				
1422	Q9UK12 ZN222_HUMAN	263	268	GksfCL				
1423	-	488	493	GwasCL				
1424 1425		572	577 194	GwasCL				
1426	Q9UK99 FBX3_HUMAN Q9UKB1 FBW1B_HUMAN	189 281	286	GlkyCL GsvlCL				
1427	Q9UKP4 ATS7_HUMAN	443	448	GwglCL				
1428	Q9UKP5 ATS6_HUMAN	545	550	GgkyCL				
1429		500	505	GkghCL				
1430	Q9UKU0 ACSL6_HUMAN	104	109	GngpCL				
1431	Q9UL25 RAB21_HUMAN	121	126	GneiCL				
1432	Q9ULB1 NRX1A_HUMAN	1048	1053	GfqgCL				
1433	Q9ULL4 PLXB3_HUMAN	1191	1196	GrgeCL				
1434	· –	1496	1501	GtvpCL				
1435		1228	1233	GgfrCL				
1436 1437	Q9UM82 SPAT2_HUMAN Q9UMF0 ICAM5_HUMAN	37 879	42 884	GsdeCL GeavCL				
1438	Q9UMW8 UBP18_HUMAN	61	66	GeaveL GqtcCL				
1439		467	472	GhgnCL				
1440	_	525	530	GqmvCL				
1441	Q9UNI1 ELA1_HUMAN	208	213	GplhCL				
1442		421	426	GhgdCL				
1443	Q9UP79 ATS8_HUMAN	562	567	GgryCL				
	Q9UP95 S12A4_HUMAN	622	627	GmslCL				
1445		1765	1770	GspvCL				
1446		881	886	GiheCL				
1447	Q9UQ05 KCNH4_HUMAN	213	218	GgsrCL				
1448 1449	Q9UQ49 NEUR3_HUMAN Q9UQ52 CNTN6_HUMAN	380 96	385	GlfgCL				
1450	Q9UQD0 SCN8A_HUMAN	949	101 954	GmyqCL GgamCL				
1451	Q9Y219 JAG2_HUMAN	907	912	GwkpCL				
1452	Q9Y236 OSGI2_HUMAN	480	485	GvtrCL				
1453	Q9Y263 PLAP_HUMAN	721	726	GkaqCL				
1454	Q9Y278 OST2_HUMAN	51	56	GaprCL				
1455	Q9Y297 FBW1A_HUMAN	344	349	GsvlCL				
1456	Q9Y2H6 FNDC3_HUMAN	790	795	GivtCL				
1457	Q9Y2L6 FRM4B_HUMAN	871	876	GsqrCL				
1458	Q9Y2P5 S27A5_HUMAN	345	350	GilgCL				
1459 1460	Q9Y2P5 S27A5_HUMAN	452 132	457 137	GkmsCL GlnqCL				
1461	Q9Y2Q1 ZN257_HUMAN Q9Y2T5 GPR52_HUMAN	205	210	GfivCL				
1462	Q9Y385 UB2J1_HUMAN	87	92	GkkiCL				
1463	Q9Y3B6 CN122_HUMAN	38	43	GeclCL				
1464		112	117	GgkiCL				
1465	Q9Y3I1 FBX7_HUMAN	71	76	GdliCL				
1466	Q9Y3N9 OR2W1_HUMAN	108	113	GsveCL				
1467	Q9Y3R4 NEUR2_HUMAN	160	165	GpghCL				
1468		182	187	GqhsCL				
	Q9Y485 DMXL1_HUMAN	187	192	GkddCL				
	Q9Y485 DMXL1_HUMAN Q9Y493 ZAN_HUMAN	2862	2867 1157	XrnvCL GtatCL				
1472	Q9Y4C0 NRX3A_HUMAN	1152 1014	1019	GfqgCL				
1473	Q9Y4F1 FARP1_HUMAN	820	825	GvphCL				
1474		1473	1478	GhypCL				
1475	Q9Y4W6 AFG32_HUMAN	31	36	GeqpCL				
1476	Q9Y535 RPC8_HUMAN	43	48	GleiCL				
1477	Q9Y561 LRP12_HUMAN	241	246	GnidCL				
1478	Q9Y574 ASB4_HUMAN	86	91	GhveCL				
1479	Q9Y575 ASB3_HUMAN	291	296	GhedCL				
1480		729	734	GtcaCL				
1481	Q9Y5J3 HEY1_HUMAN	126 45	131	GfreCL GveiCL				
1482 1483	Q9Y5N5 HEMK2_HUMAN Q9Y5Q5 CORIN_HUMAN	43 424	50 429	GveiCL GdqrCL				
	Q9Y5R5 DMRT2_HUMAN	130	135	GuqrCL GvvsCL				
1485	-	153	158	GsnpCL				
1486	_	1374	1379	GsvqCL				
1487	-	73	78	GvpkCL				
1488	Q9Y616 IRAK3_HUMAN	395	400	GldsCL				
1489	Q9Y644 RFNG_HUMAN	203	208	GagfCL				
1490	Q9Y662 OST3B_HUMAN	7	12	GgrsCL				

	#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
10	1491	Q9Y666 S12A7_HUMAN	622	627	GmslCL
	1492	Q9Y6H5 SNCAP_HUMAN	361	366	GhaeCL
	1493	Q9Y6I4 UBP3_HUMAN	449	454	GpesCL
	1494	Q9Y6N6 LAMC3_HUMAN	885	890	GqcsCL
	1495	Q9Y6R1 S4A4_HUMAN	512	517	GaifCL
	1496	Q9Y6R7 FCGBP_HUMAN	1661	1666	GqgvCL
15	1497	Q9Y6R7 FCGBP_HUMAN	2388	2393	GqcgCL
13	1498	Q9Y6R7 FCGBP_HUMAN	2862	2867	GqgvCL
	1499	Q9Y6R7 FCGBP_HUMAN	3589	3594	GqcgCL
	1500	Q9Y6R7 FCGBP_HUMAN	4063	4068	GqgvCL
	1501	Q9Y6R7 FCGBP_HUMAN	4790	4795	GqcgCL
	1502	Q9Y6R7 FCGBP_HUMAN	4852	4857	GcgrCL
20	1503	Q9Y6R7 FCGBP_HUMAN	5032	5037	GcpvCL

These peptides are likely to have anti-angiogenic activity. Methods for testing for such activity are described herein.

### Example 4

### Collagen Derived Peptides

The same procedure as used for the C-X-C chemokines can be repeated for the case of the collagen related fragments. Because the number of the experimentally tested peptides is small in the calculation, all the theoretically predicted fragments are considered. Both the short and long predicted fragments are introduced. Two predominant motifs were calculated. One of them is the most abundant and is characterized 35 by a conserved 4-amino acid repeat. It can be described by the following generic sequence: C-N-X3-V-C (SEQ ID NO: 2288) (FIG. 6A). This motif can be localized either upstream or downstream of the peptide sequence. If the peptides are separated according to the location of the C-N-X3-V-C motif (SEQ ID NO: 2288), it can be either near the amino or carboxy terminal of the peptide. This provides for the identification of a set of two more definitive motifs (FIGS. 6B and 6C). The 4-letter motif appears upstream is the C-N-X3-V-C-X2-A-X-R-N-D-X-S-Y-W-L (SEQ ID NO: 2315) (FIG. 45 **6**B), whereas the motif that appears downstream is the L-X2-F-S-T-X-P-F-X2-C-N-X3-V-C (SEQ ID NO: 2316) (FIG.

Apart from the aforementioned 7-mer there is another motif that is present in a smaller subset of collagen derived 50 peptides. Those peptides do not include the C-N-X3-V-C (SEQ ID NO: 2288). This motif is described by the generic sequence X2-P-F-X-E-C-X-G-X8-A-N (SEQ ID NO: 2317). Common modifications can be described by the sequence X2-P-F-(I/L)-E-C-X-G-X-(R/G)-X-(Y/F)-(Y/F)-A-N (SEQ 55 ID NO: 2318) (FIG. 7).

If only the short identified anti-angiogenic fragments are considered then the multiple alignment algorithm may be used to identify motifs present only within this subset of the peptides. The alignment is shown in FIG. 8. These motifs are similar to those identified herein. A more generic 3-common letter motif, the P-F-X2-C motif can be distinguished.

In the case of collagens two generic motifs were identified. The first one is the C-N-X3-V-C (SEQ ID NO: 2288). Using this motif as a query and scanning the Prosite database 24 hits 65 in 24 different proteins were identified. These candidate antiangiogenic peptides are listed in Table 5 (SEQ ID NO: 1504-1527).

Accession

Number|Protein Name

1555 O75419|CC45L\_HUMAN

1556 O75473|LGR5\_HUMAN

1558 O75581|LRP6\_HUMAN

1559 O75794|CD123\_HUMAN

1560 O75882|ATRN\_HUMAN

1561 O76031|CLPX\_HUMAN

1562 O95006|OR2F2\_HUMAN

1563 O95007|OR6B1\_HUMAN

1565 O95202|LETM1\_HUMAN

1564 O95149|SPN1\_HUMAN

1566 O95409|ZIC2 HUMAN

1567 O95450|ATS2\_HUMAN

1568 O95759|TBCD8 HUMAN

1569 O95841|ANGL1\_HUMAN

1570 O95886|DLGP3 HUMAN

1571 P02461|CO3A1\_HUMAN

1572 P02462|CO4A1\_HUMAN

1573 P02462|CO4A1\_HUMAN

1575 P08572|CO4A2\_HUMAN

1576 P08572|CO4A2\_HUMAN

1577 P08581|MET\_HUMAN

1578 P09172|DOPO\_HUMAN

1579 P0C0L4|CO4A\_HUMAN

1580 P0C0L5|CO4B HUMAN

1581 P15309|PPAP\_HUMAN

1583 P18084|ITB5\_HUMAN

1582 P17021|ZNF17\_HUMAN

1584 P20645|MPRD\_HUMAN

1585 P20851|C4BB\_HUMAN

1586 P20933|ASPG\_HUMAN

1587 P21673|SAT1\_HUMAN

1588 P21854|CD72\_HUMAN

1589 P22309|UD11\_HUMAN

1590 P22362|CCL1\_HUMAN

1591 P22681|CBL\_HUMAN

1574 P08151|GLI1 HUMAN

1557 O75478|TAD2L\_HUMAN

1554 O75197|LRP5\_HUMAN

## TABLE 6-continued

First

Amino

acid

547

38

304

147

969

313

93

285

195

51

336

569

67

276

98

80

1501

1612

1545

1654

534

136

731

731

157

350

546

130

50

222

152

29

417

210

3083

483

252

117

439

869

1517

1628

244

153

26

252

205

258

149

57

538

650

141

528

405

471

79

315

273

376

195

581

1411

173

Last Amino

acid

551

42

308

151

973

317

97

289

199

55

340

573

71

280 102

84

1505

1616

177

1549

1658

538

140

735

735

161

354

550

134

17

54

226

156

33

421

214

3087

487

121

443

873

1521

1632

248

157

30

256

209

262

153

61

542

654

145

532

1415

409

475

83

319

277

380

199

585

Sequence

PFytC

PFlyC

PFkpC

PFfIC

PFyqC

PFihC

PFgqC

PFaiC

PFqsC

PFivC

PFydC

PFgcC

PFpgC

PFgsC

PFsrC

PFkdC

PFdtC

PFgeC

PFIfC

PFieC

PFptC

PFlyC

PFieC

PFvqC

PFgtC

PFlsC

PFlsC

PFrnC

PFycC

PFceC

PFysC

PFpiC

PFIIC

PFyhC

PFftC

PFlpC

PFsrC

PFcrC

PFscC PFegC

PFrgC

PFqlC

PFivC PFdgC

PFvqC PFffC

PFdyC

PFfkC

PFmfC

PFieC

PFehC

PFhlC

PFehC

PFivC

PFyvC

PFe<sub>2</sub>C

PFdnC

PFlkC

PFsnC

PFldC

PFpvC

PFssC

PFyrC

PFenC

PFpsC

PFleC

PFlyC

PFvlC

PFkgC

PFpfC

PFcsC

84

#### Collagens Motif: P-F-X2-C Number of Locations: 306 Number of Different Proteins: 288

Collagens
Motif: C-N-X(3)-V-C (SEQ ID NO: 2288)
Number of Locations: 24
Number of Different Proteins: 24

#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence	
1504	O14514 BAI1_HUMAN	400	406	CNnsaVC	- :
	O75093 SLIT1_HUMAN	507	513	CNsdvVC	
1506	O75534 CSDE1_HUMAN	733	739	CNvwrVC	
1507	P02462 CO4A1_HUMAN	1505	1511	CNinnVC	
1508	P08572 CO4A2_HUMAN	1549	1555	CNpgdVC	
1509	P09758 TACD2_HUMAN	119	125	CNqtsVC	
1510	P25391 LAMA1_HUMAN	751	757	CNvhgVC	
1511	P29400 CO4A5_HUMAN	1521	1527	CNinnVC	
1512	P53420 CO4A4_HUMAN	1525	1531	CNihqVC	
1513	P83110 HTRA3_HUMAN	48	54	CNcclVC	
1514	Q01955 CO4A3_HUMAN	1505	1511	CNvndVC	
1515	Q13625 ASPP2_HUMAN	1002	1008	CNnvqVC	
1516	Q13751 LAMB3_HUMAN	572	578	CNrypVC	
1517	Q14031 CO4A6_HUMAN	1527	1533	CNineVC	
1518	Q8WWQ8 STAB2_HUMAN	1970	1976	CNnrgVC	
1519	Q96GX1 TECT2_HUMAN	642	648	CNrneVC	
1520	Q99965 ADAM2_HUMAN	621	627	CNdrgVC	
1521	Q9BX93 PG12B_HUMAN	112	118	CNqldVC	
1522	Q9BYD5 CNFN_HUMAN	32	38	CNdmpVC	
1523	Q9H013 ADA19_HUMAN	659	665	CNghgVC	2
1524	Q9HBG6 IF122_HUMAN	436	442	CNIIvVC	
1525	Q9P2R7 SUCB1_HUMAN	152	158	CNqvIVC	
1526	Q9UBX1 CATF_HUMAN	89	95	CNdpmVC	
1527	Q9UKF2 ADA30_HUMAN	638	644	CNtrgVC	_

The second motif is the P-F-X2-C. Again using this motif as a query at the Prosite 306 locations that contain the specific amino acid sequence were identified in 288 different proteins. The hits included peptides shown in Table 6 (SEQ ID Nos: 1528-1833).

TABLE 6

Collagens	
Motif: P-F-X2-C	
Number of Locations: 306	
Number of Different Proteins: 288	

	Motif: P-	40	1331 122001 CDL_HOMAIN			
	Number of Lo	40	1592 P23942 RDS_HUMAN			
	Number of Differ		1593 P24043 LAMA2_HUMAN			
		-	1594 P24043 LAMA2_HUMAN			
		First	Last			1595 P24903 CP2F1_HUMAN
	Accession	Amino	Amino			1596 P25098 ARBK1_HUMAN
#	Number Protein Name	acid	acid	Sequence		1597 P25490 TYY1_HUMAN
	Transer i Totelli Transe		uera	Bequence	45	1598 P25929 NPY1R_HUMAN
1528	O00116 ADAS HUMAN	561	565	PFstC		1599 P26718 NKG2D_HUMAN
1529	O00182 LEG9 HUMAN	98	102	PFdlC		1600 P26927 HGFL_HUMAN
1530	O00206 TLR4_HUMAN	702	706	PFqlC		1601 P27987 IP3KB_HUMAN
	O00270 GPR31 HUMAN	2	6	PFpnC		1602 P29400 CO4A5_HUMAN
	O00398 P2Y10 HUMAN	288	292	PFclC		1603 P29400 CO4A5_HUMAN
	O00507 USP9Y HUMAN	259	263	PFgqC	50	1604 P34896 GLYC_HUMAN
	O14646 CHD1_HUMAN	450	454	PFkdC		1605 P35504 UD15_HUMAN
	O14843 FFAR3 HUMAN	84	88	PFilC		1606 P35523 CLCN1_HUMAN
	O14978 ZN263 HUMAN	547	551	PFseC		1607 P35626 ARBK2 HUMAN
	O15015 ZN646 HUMAN	880	884	PFlcC		1608 P36383 CXA7 HUMAN
	O15031 PLXB2 HUMAN	611	615	PFydC		1609 P36508 ZNF76_HUMAN
	O15037 K0323_HUMAN	423	427	PFtlC	55	1610 P36509 UD12_HUMAN
	O15453 NBR2_HUMAN	9	13	PFlpC	33	1611 P36894 BMR1A HUMAN
	O15529 GPR42 HUMAN	84	88	PFilC		1612 P41180 CASR_HUMAN
	O43556 SGCE HUMAN	207	211	PFssC		1613 P42338 PK3CB HUMAN
	O60299 K0552_HUMAN	308	312	PFaaC		1614 P42575 CASP2 HUMAN
	O60343 TBCD4_HUMAN	89	93	PFlrC		1615 P45974 UBP5 HUMAN
	O60431 OR1I1 HUMAN	93	97	PFvgC		1616 P46531 NOTC1 HUMAN
	O60449 LY75 HUMAN	1250	1254	PFqnC	60	1617 P48637 GSHB_HUMAN
	O60481 ZIC3_HUMAN	331	335	PFpgC		1618 P49257 LMAN1 HUMAN
	O60486 PLXC1_HUMAN	618	622	PFtaC		1619 P49888 ST1E1 HUMAN
	O60494 CUBN HUMAN	3302	3306	PFsiC		1620 P50052 AGTR2_HUMAN
	O60603 TLR2_HUMAN	669	673	PFklC		1621 P50876 UB7I4_HUMAN
	O60656 UD19 HUMAN	149	153	PFdnC		1622 P51606 RENBP HUMAN
	O60706 ABCC9 HUMAN	627	631	PFesC	65	1623 P51617 IRAK1_HUMAN
	O75152 ZC11A HUMAN	23	27	PFrhC		1624 P51689 ARSD HUMAN
1555	G/3132/ZCITA_HOWAN	23	21	TTIIC		1024 131009 AKSD_HUMAN

TABLE 6-continued

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TABLE 0-0	Commueu			-	TABLE 0-C	ommueu			
Collag	gens				Collag	ens			
Motif: P-	F-X2-C				Motif: P-F-X2-C				
Number of Lo Number of Differe		288		5	Number of Lo Number of Differe		288		
Number of Difference	mi Frotenis	200		-	Number of Differe	nt Flotenis. 2	200		
	First	Last				First	Last		
Accession	Amino	Amino	G.		Accession	Amino	Amino	a	
# Number Protein Name	acid	acid	Sequence	_	# Number Protein Name	acid	acid	Sequence	
1625 P51690 ARSE_HUMAN	576	580	PFplC	10	1696 Q86SQ6 GP123_HUMAN	863	867	PFiiC	
1626 P52740 ZN132_HUMAN	369	373	PFecC		1697 Q86T65 DAAM2_HUMAN	548	552	PFacC	
1627 P52747 ZN143_HUMAN	318	322	PFegC		1698 Q86V97 KBTB6_HUMAN	355	359	PFIcC	
1628 P53420 CO4A4_HUMAN 1629 P53420 CO4A4_HUMAN	1521 1630	1525 1634	PFayC PFleC		1699 Q86XI2 CNDG2_HUMAN 1700 Q86YT6 MIB1_HUMAN	1043 909	1047 913	PFsrC PFimC	
1630 P53621 COPA_HUMAN	1165	1169	PFdiC		1701 Q8IUH2 CREG2_HUMAN	152	156	PFgnC	
1631 P54198 HIRA_HUMAN	215	219	PFdeC	15	1702 Q8IWU5 SULF2_HUMAN	745	749	PFcaC	
1632 P54793 ARSF_HUMAN	570	574	PFclC	13	1703 Q8IWV8 UBR2_HUMAN	1514	1518	PFlkC	
1633 P54802 ANAG_HUMAN	401	405	PFiwC		1704 Q8IWX5 SGPP2_HUMAN	257	261	PFfIC	
1634 P55157 MTP_HUMAN	823 183	827 187	PFlvC PFccC		1705 Q8IX07 FOG1_HUMAN	293 287	297	PFpqC PFplC	
1635 P62079 TSN5_HUMAN 1636 P78357 CNTP1_HUMAN	926	930	PFscC PFvgC		1706 Q8IX29 FBX16_HUMAN 1707 Q8IXT2 DMRTD_HUMAN	224	291 228	PFplC PFttC	
1637 P78527 PRKDC_HUMAN	2853	2857	PFvsC		1708 Q8IZF5 GP113_HUMAN	62	66	PFpaC	
1638 P81133 SIM1_HUMAN	200	204	PFdgC	20	1709 Q8IZQ8 MYCD_HUMAN	403	407	PFqdC	
1639 P98088 MUC5A_HUMAN	290	294	PFkmC		1710 Q8IZW8 TENS4_HUMAN	423	427	PFttC	
1640 Q01955 CO4A3_HUMAN	1501	1505	PFlfC		1711 Q8N0W3 FUK_HUMAN	100	104	PFddC	
1641 Q01955 CO4A3_HUMAN 1642 Q02817 MUC2_HUMAN	1612 597	1616 601	PFleC PFgrC		1712 Q8N122 RPTOR_HUMAN 1713 Q8N1G1 REXO1_HUMAN	1033 278	1037 282	PFtpC PFgsC	
1643 Q02817 MUC2_HUMAN	1375	1379	PFglC		1714 Q8N1G2 K0082_HUMAN	790	794	PFhiC	
1644 Q02817 MUC2_HUMAN	4916	4920	PFywC	25	1715 Q8N201 INT1_HUMAN	1573	1577	PFpaC	
1645 Q03395 ROM1_HUMAN	213	217	PFscC		1716 Q8N475 FSTL5_HUMAN	61	65	PFgsC	
1646 Q07912 ACK1_HUMAN	293	297	PFawC		1717 Q8N567 ZCHC9_HUMAN	182	186	PFakC	
1647 Q12830 BPTF_HUMAN	2873	2877	PFyqC		1718 Q8N7R0 NANG2_HUMAN	166	170	PFynC	
1648 Q12836 ZP4_HUMAN 1649 Q12866 MERTK_HUMAN	238 313	242 317	PFtsC PFrnC		1719 Q8N8U9 BMPER_HUMAN 1720 Q8N9L1 ZIC4_HUMAN	234 207	238 211	PFgsC PFpgC	
1650 Q12950 FOXD4_HUMAN	291	295	PFpcC	30	1721 Q8NB16 MLKL_HUMAN	411	415	PFqgC	
1651 Q12968 NFAC3_HUMAN	327	331	PFqyC	50	1722 Q8NG11 TSN14_HUMAN	183	187	PFscC	
1652 Q13191 CBLB_HUMAN	409	413	PFcrC		1723 Q8NGC3 O10G2_HUMAN	98	102	PFggC	
1653 Q13258 PD2R_HUMAN	4	8	PFyrC		1724 Q8NGC4 O10G3_HUMAN	94	98	PFggC	
1654 Q13356 PPIL2_HUMAN 1655 Q13607 OR2F1_HUMAN	38 93	42 97	PFdhC PFqsC		1725 Q8NGJ1 OR4D6_HUMAN 1726 Q8NH69 OR5W2_HUMAN	165 93	169 97	PFpfC PFvcC	
1656 Q13753 LAMC2_HUMAN	409	413	PFgtC	2.5	1720 Q8NH85 OR5R1_HUMAN	93	97	PFygC PFhaC	
1657 Q13936 CAC1C_HUMAN	2179	2183	PFvnC	35	1728 Q8NHU2 CT026_HUMAN	442	446	PFntC	
1658 Q14031 CO4A6_HUMAN	1523	1527	PFiyC		1729 Q8NHY3 GA2L2_HUMAN	359	363	PFlrC	
1659 Q14031 CO4A6_HUMAN	1632	1636	PFieC		1730 Q8NI51 BORIS_HUMAN	369	373	PFqcC	
1660 Q14137 BOP1_HUMAN 1661 Q14330 GPR18_HUMAN	400 247	404 251	PFptC PFhiC		1731 Q8TCB0 IFI44_HUMAN	246 88	250 92	PFilC PFelC	
1662 Q14643 ITPR1_HUMAN	526	530	PFtdC		1732 Q8TCE9 PPL13_HUMAN 1733 Q8TCT7 PSL1_HUMAN	275	279	PFgkC	
1663 Q15042 RB3GP_HUMAN	267	271	PFgaC	40	1734 Q8TD94 KLF14_HUMAN	198	202	PFpgC	
1664 Q15389 ANGP1_HUMAN	282	286	PFrdC		1735 Q8TF76 HASP_HUMAN	474	478	PFshC	
1665 Q15583 TGIF_HUMAN	269	273	PFhsC		1736 Q8WW14 CJ082_HUMAN	22	26	PFlsC	
1666 Q15583 TGIF_HUMAN	314	318	PFslC		1737 Q8WW38 FOG2_HUMAN	299	303	PFpqC	
1667 Q15761 NPY5R_HUMAN 1668 Q15915 ZIC1_HUMAN	128 305	132 309	PFlqC PFpgC		1738 Q8WWG1 NRG4_HUMAN 1739 Q8WWZ7 ABCA5_HUMAN	32 361	36 365	PFcrC PFchC	
1669 Q16363 LAMA4_HUMAN	1788	1792	PFtgC	45	1740 Q8WXT5 FX4L4_HUMAN	295	299	PFpcC	
1670 Q16572 VACHT_HUMAN	517	521	PFdeC		1741 Q8WYR1 PI3R5_HUMAN	814	818	PFavC	
1671 Q16586 SGCA_HUMAN	205	209	PFstC		1742 Q8WZ42 TITIN_HUMAN	31091	31095	PFpiC	
1672 Q16773 KAT1_HUMAN	123	127	PFfdC		1743 Q8WZ60 KLHL6_HUMAN	432	436	PFhnC	
1673 Q16878 CDO1_HUMAN 1674 Q2TBC4 CF049_HUMAN	160 298	164 302	PFdtC PFstC		1744 Q92485 ASM3B_HUMAN 1745 Q92793 CBP_HUMAN	41 1279	45 1283	PFqvC PFvdC	
1675 Q49AM1 MTER3_HUMAN	28	32	PFlaC	50	1746 Q92838 EDA_HUMAN	328	332	PFlqC	
1676 Q53FE4 CD017_HUMAN	77	81	PFanC	50	1747 Q92995 UBP13_HUMAN	540	544	PFsaC	
1677 Q53G59 KLH12_HUMAN	240	244	PFirC		1748 Q93008 USP9X_HUMAN	251	255	PFgqC	
1678 Q53T03 RBP22_HUMAN	517	521	PFpvC		1749 Q96F10 SAT2_HUMAN	50	54	PFyhC	
1679 Q5IJ48 CRUM2_HUMAN	762	766 245	PFrgC		1750 Q96FV3 TSN17_HUMAN	185	189	PFscC	
1680 Q5T442 CXA12_HUMAN 1681 Q5VYX0 RENAL_HUMAN	241 310	314	PFfpC PFlaC		1751 Q96IK0 TM101_HUMAN 1752 Q96L50 LLR1_HUMAN	27 344	31 348	PFwgC PFhlC	
1682 Q5W0N0 CI057_HUMAN	89	93	PFhgC	55	1753 Q96L73 NSD1_HUMAN	456	460	PFedC	
1683 Q6NSW7 NANP8_HUMAN	239	243	PFynC		1754 Q96P88 GNRR2_HUMAN	184	188	PFtqC	
1684 Q6P2Q9 PRP8_HUMAN	1892	1896	PFqaC		1755 Q96PZ7 CSMD1_HUMAN	2139	2143	PFprC	
1685 Q6PRD1 GP179_HUMAN	232	236	PFleC		1756 Q96R06 SPAG5_HUMAN	378 542	382	PFstC	
1686 Q6TCH4 PAQR6_HUMAN 1687 Q6UB98 ANR12_HUMAN	95 1949	99 1953	PFasC PFsaC		1757 Q96RG2 PASK_HUMAN 1758 Q96RJ0 TAAR1_HUMAN	542 266	546 270	PFasC PFfiC	
1688 Q6UB99 ANR11_HUMAN	2552	2556	PFsaC	60	1759 Q96RQ9 OXLA_HUMAN	32	36	PFekC	
1689 Q6UXZ4 UNC5D_HUMAN	766	770	PFtaC		1760 Q96SE7 ZN347_HUMAN	798	802	PFsiC	
1690 Q7Z434 MAVS_HUMAN	431	435	PFsgC		1761 Q96T25 ZIC5_HUMAN	470	474	PFpgC	
1691 Q7Z6J6 FRMD5_HUMAN	87	91	PFtmC		1762 Q99666 RGPD8_HUMAN	517	521	PFpvC	
1692 Q7Z7G8 VP13B_HUMAN 1693 Q7Z7G8 VP13B_HUMAN	441 1423	445 1427	PFfdC PFrnC		1763 Q99698 LYST_HUMAN 1764 Q99726 ZNT3_HUMAN	254 51	258 55	PFdlC PFhhC	
1694 Q7Z7M1 GP144_HUMAN	352	356	PFIcC	65	1765 Q9BSE5 SPEB_HUMAN	204	208	PFrrC	
1695 Q86SJ6 DSG4_HUMAN	523	527	PFtfC		1766 Q9BWQ6 YIPF2_HUMAN	124	128	PFwiC	
			-					=	

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### Collagens Motif: P-F-X2-C

#### Motif: P-F-X2-C Number of Locations: 306 Number of Different Proteins: 288

		First	Last	
	Accession	Amino	Amino	
#	Number Protein Name	acid	acid	Sequence
	Q9BXC9 BBS2_HUMAN	530	534	PFqvC
	Q9BXJ4 C1QT3_HUMAN	18	22	PFclC
	Q9BXK1 KLF16_HUMAN Q9BZE2 PUS3_HUMAN	130 261	134 265	PFpdC PFqlC
1771	Q9C0C4 SEM4C_HUMAN	719	723	PFrpC
	Q9C0E2 XPO4_HUMAN	50	54	PFavC
	Q9C0I4 THS7B_HUMAN	1482	1486	PFsyC
	Q9GZN6 S6A16_HUMAN Q9GZU2 PEG3_HUMAN	271 1330	275 1334	PFflC PFyeC
	O9GZZ0/HXD1 HUMAN	162	166	PFpaC
1777	Q9H0A6 RNF32_HUMAN	344	348	PFhaC
	Q9H0B3 K1683_HUMAN	326	330	PFqiC
	Q9H267 VP33B_HUMAN Q9H2J1 CI037_HUMAN	189 102	193 106	PFpnC PFekC
	Q9H3H5 GPT_HUMAN	77	81	PFlnC
	Q9H8V3 ECT2_HUMAN	239	243	PFqdC
	Q9H9S0 NANOG_HUMAN	239	243	PFynC
	Q9H9V4 RN122_HUMAN Q9HAQ2 KIF9_HUMAN	3 291	7 295	PFqwC PFrqC
	Q9HAW7 UD17_HUMAN	149	153	PFdaC
	Q9HAW8 UD110_HUMAN	149	153	PFdtC
	Q9HAW9 UD18_HUMAN	149	153	PFdaC
	Q9HBX8 LGR6_HUMAN	412	416	PFkpC
	Q9NQW8 CNGB3_HUMAN O9NRZ9 HELLS HUMAN	309 273	313 277	PFdiC PFlvC
	Q9NTG7 SIRT3_HUMAN	30	34	PFqaC
1793	Q9NWZ5 UCKL1_HUMAN	370	374	PFqdC
	Q9NY30 BTG4_HUMAN	98	102	PFevC
	Q9NYM4 GPR83_HUMAN Q9NYV6 RRN3_HUMAN	342 561	346 565	PFiyC PFdpC
	Q9NYW1 TA2R9_HUMAN	190	194	PFilC
	Q9NYW3 TA2R7_HUMAN	193	197	PFcvC
	Q9NZ56 FMN2_HUMAN	716	720	PFsdC
	Q9NZ71 RTEL1_HUMAN	495	499	PFpvC
	Q9NZD2 GLTP_HUMAN Q9P2N4 ATS9_HUMAN	31 596	35 600	PFfdC PFgtC
	Q9UBR1 BUP1_HUMAN	124	128	PFafC
	Q9UBS0 KS6B2_HUMAN	344	348	PFrpC
	Q9UET6 RRMJ1_HUMAN	234	238	PFvtC
	Q9UHD4 CIDEB_HUMAN Q9UKA4 AKA11_HUMAN	37 917	41 921	PFrvC PFshC
	Q9ULC3 RAB23_HUMAN	230	234	PFssC
	Q9ULJ3 ZN295_HUMAN	125	129	PFptC
	Q9ULK4 CRSP3_HUMAN	1086	1090	PFpnC
	Q9ULL4 PLXB3_HUMAN Q9ULV8 CBLC_HUMAN	24 387	28 391	PFglC PFcrC
	Q9UM47 NOTC3_HUMAN	1357	1361	PFfrC
1814	Q9UNQ2 DIMT1_HUMAN	146	150	PFfrC
	Q9Y3D5 RT18C_HUMAN	86	90	PFtgC
	Q9Y3F1 TA6P_HUMAN Q9Y3R5 CU005_HUMAN	25 255	29 259	PFpsC PFytC
	Q9Y450 HBS1L_HUMAN	487	491	PFrlC
	Q9Y493 ZAN_HUMAN	1364	1368	PFetC
	Q9Y493 ZAN_HUMAN	1751	1755	PFsqC
1821	Q9Y493 ZAN_HUMAN Q9Y548 YIPF1_HUMAN	2556 123	2560 127	PFaaC PFwiC
	Q9Y5L3 ENP2_HUMAN	324	328	PFsrC
	Q9Y5P8 2ACC_HUMAN	272	276	PFqdC
	Q9Y664 KPTN_HUMAN	143	147	PFqlC
	Q9Y678 COPG_HUMAN	226	230	PFayC
	Q9Y6E0 STK24_HUMAN O9Y6R7 FCGBP_HUMAN	371 683	375 687	PFsqC PFavC
	Q9Y6R7 FCGBP_HUMAN	1074	1078	PFreC
1830	Q9Y6R7 FCGBP_HUMAN	1888	1892	PFttC
	Q9Y6R7 FCGBP_HUMAN	3089	3093	PFttC
	Q9Y6R7 FCGBP_HUMAN Q9Y6R7 FCGBP_HUMAN	4290 5059	4294 5063	PFttC PFatC
1000	2. Total TODI TIOMAN	2029	5005	11410

Finally the motifs that are found within the predicted pep- 6 tides that are derived from tissue inhibitors of metalloprotein-ases were calculated. Because of the small number of pep-

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tides present in the peptide pool the loop-6 fragment of TIMP-2 was also included in the calculation. This loop is known to have anti-angiogenic activity. For this case the common motif among the peptide sequences is the E-C-L-W-X-D-X8-G-X-Y-X5-C (SEQ ID NO: 2319) as shown in the FIG 9

### Example 5

### Novel Peptides from the Somatotropin and Serpin Protein Families

Growth Hormone (GH) and prolactin proteins contain a somatotropin conserved domain. Pigment epithelium derived factor (PEDF) contains a serpin conserved domain. There are a number of short peptides, smaller than 25 amino acids, from these two protein families that can be used to identify sequences having similarity to these peptides within the human proteome. Such peptides include the recently identified short fragments of GH and prolactin (Nguyen et al., (2006) *Proc Natl Acad Sci USA* 103, 14319-14324), and short fragments of PEDF (Filleur et al., (2005) *Cancer Res* 65, 5144-5152).

After searching within the human proteome for similar sequences to those of the short peptides and filtering the results for only the statistically significant similarities using a Monte Carlo algorithm eleven novel similar peptides were identified, eight similar to the short fragment derived from growth hormone (FIG. 10A) and three from the short fragment of PEDF (FIG. 10B). These sequences are also included in Tables 7A and 7B.

TABLE 7A

Table of	the	amin	acid	sequ	iences	of	the	peptides
I	redi	cted :	similar	to	Growth	ı Ho	ormoi	ne

Protein Name	Peptide Location	Peptide sequence
Placental	AAA98621	LLRISLLLIESWLE
Lactogen	(101-114)	(SEQ ID NO: 2291)
hGH-V	AAB59548	LLRISLLLTQSWLE
	(101-114)	(SEQ ID NO: 2292)
GH2	CAG46722	LLHISLLLIOSWLE
	(101-114)	(SEQ ID NO: 2293)
Chorionic	AAA52116	LLRLLLLIESWLE
somatomammotropin	(101-113)	
Chorionic	AAI19748	LLHISLLLIESRLE
somatomammotropin	(12-25)	(SEQ ID NO: 2295)
hormone-like 1		
Transmembrane	NP_060474	LLRSSLILLQGSWF
protein 45A	 (181-194)	(SEQ ID NO: 2296)
IL-17 receptor C	Q8NAC3	RLRLLTLQSWLL
-	(376-387)	(SEQ ID NO: 2297)
Neuropeptide FF	Q9Y5X5	LLIVALLFILSWL
receptor 2	(378-390)	(SEQ ID NO: 2298)
Brush border	AAC27437	LMRKSOILIS SWF
myosin-I	(719-731)	(SEQ ID NO: 2299)

Peptide sequence

(SEQ ID NO: 2300)

(SEQ ID NO: 2301)

EIELVEEEPPF

AEDLLSEEDPF (SEQ ID NO: 2302)

TLDLIQEEDPS (SEQ ID NO: 2303)

the peptides predicted similar to PEDF

Peptide Location

CAD32371 (67-77)

CAI14263 (66-76)

AAH47327

(438 - 448)

Protein Name

polypeptide 8

Caspase 10

CKIP-1

90 TABLE 8-continued

Amino acid sequences	of peptides that contain	the somatotropin motif.
	Somatotropins	

Motif: L-X(3)-L-L-X(3)-S-X-L (SEQ ID NO: 2289) Number of Locations: 139 Number of Different Proteins: 139

		Number of Differ	ent Prote	ins: 139	
			First		
			Ami-	Last	
		Accession	no	Amino	
10	#	Number Protein Name	acid	acid	Sequence
	4055	D40000017777777		= 60	- 1
		P19838 NFKB1_HUMAN	558	569	LvrdLLevtSgL
		P22079 PERL_HUMAN	512	523	LvrgLLakkSkL
		P23276 KELL_HUMAN	53	64	LilgLLlcfSvL
		P24394 IL4RA_HUMAN	4 5	15	LesgLLfpvScL
15		P29320 EPHA3_HUMAN P31512 FMO4_HUMAN	524	16 535	LsilLLlscSvL LaslLLickSsL
	1861	P35270 SPRE_HUMAN	26	333	LlasLLspgSvL
	1862		20	31	LpprLLarpSlL
		P42575 CASP2_HUMAN	114	125	LedmLLttlSgL
		P46721 SO1A2_HUMAN	396	407	LleyLLyflSfL
	1865		201	212	LnskLLdirSyL
20		P59531 T2R12_HUMAN	188	199	LisfLLsliSlL
		P69849 NOMO3_HUMAN	1180	1191	LiplLLqltSrL
		P98161 PKD1_HUMAN	82	93	LdvgLLanlSaL
		P98171 RHG04_HUMAN	153	164	LqdeLLevvSeL
		P98196 AT11A_HUMAN	1077	1088	LaivLLvtiSlL
	1871	Q08431 MFGM_HUMAN	10	21	LcgaLLcapSlL
25	1872	Q08AF3 SLFN5_HUMAN	533	544	LvivLLgfkSfL
	1873	Q12952 FOXL1_HUMAN	293	304	LgasLLaasSsL
	1874	Q13275 SEM3F_HUMAN	2	13	LvagLLlwaSlL
	1875	Q13394 MB211_HUMAN	300	311	LngiLLqliScL
	1876	Q13609 DNSL3_HUMAN	8	19	LlllLLsihSaL
	1877	Q13619 CUL4A_HUMAN	213	224	LlrsLLgmlSdL
30	1878	Q13620 CUL4B_HUMAN	349	360	LlrsLLsmlSdL
	1879	Q14406 CSHL_HUMAN	84	95	LhisLLlieSrL
	1880		8	19	LlvlLLvalSaL
	1881	Q15155 NOMO1_HUMAN	1180	1191	LiplLLqltSrL
	1882	Q15760 GPR19_HUMAN	279	290	LilnLLfllSwL
	1883	Q53RE8 ANR39_HUMAN	166	177	LacdLLpcnSdL
35	1885	Q5FWE3 PRRT3_HUMAN Q5GH73 XKR6_HUMAN	586 630	597 641	LatdLLstwSvL LlyeLLqyeSsL
	1886		194	205	LnraLLmtfSlL
	1887	` _	1180	1191	LiplLLqltSrL
	1888	Q5JWR5 DOP1_HUMAN	506	517	LpqlLLrmiSaL
	1889	Q5UIP0 RIF1_HUMAN	2413	2424	LsknLLaqiSaL
	1890	Q5VTE6 ANGE2_HUMAN	175	186	LsqdLLednShL
40	1891	Q5VU43 MYOME_HUMAN	1932	1943	LreaLLssrShL
	1892	Q5VYK3 ECM29_HUMAN	1296	1307	LipaLLeslSvL
	1893	Q68D06 SLN13_HUMAN	554	565	LvivLLgfrSlL
	1894	Q6GYQ0 GRIPE_HUMAN	641	652	LwddLLsvlSsL
	1895	` =	166	177	LvpwLLlgaSwL
4.5		Q6ZMH5 S39A5_HUMAN	217	228	LavlLLslpSpL
45	1897	_	532	543	LhnsLLqrkSkL
	1898	Q6ZVD8 PHLPL_HUMAN	313	324	LfpiLLceiStL
	1899 1900	Q6ZVE7 GOT1A_HUMAN	23	34 938	LfgtLLyfdSvL LrveLLsasSlL
	1900	Q70J99 UN13D_HUMAN Q7Z3Z4 PIWL4_HUMAN	927 139	150	LriaLLyshSeL
		Q7Z6Z7 HUWE1_HUMAN	841	852	LqegLLqldSiL
50		Q7Z7L1 SLN11_HUMAN	554	565	LvivLLgfrSlL
30		Q86SM5 MRGRG_HUMAN	223	234	LinfLLpvfSpL
		Q86U44 MTA70_HUMAN	78	89	LekkLLhhlSdL
		Q86UQ4 ABCAD_HUMAN	3182	3193	LlnsLLdivSsL
		Q86WI3 NLRC5_HUMAN	1485	1496	LlqsLLlslSeL
	1908	Q86YC3 LRC33_HUMAN	263	274	LffpLLpqySkL
55	1909	Q8IYK4 GT252_HUMAN	9	20	LawsLLllsSaL
55	1910	Q8IYS0 GRM1C_HUMAN	485	496	LesdLLieeSvL
	1911	Q8IZL8 PELP1_HUMAN	33	44	LrllLLesvSgL
		Q8IZY2 ABCA7_HUMAN	1746	1757	LftlLLqhrSqL
		Q8N0X7 SPG20_HUMAN	322	333	LfedLLrqmSdL
		Q8N6M3 CT142_HUMAN	33	44	LagsLLkelSpL
60		Q8N816 TMM99_HUMAN	96	107	LlpcLLgvgSwL
		Q8NBM4 PDHL1_HUMAN Q8NCG7 DGLB HUMAN	15 555	26 566	LsksLLlvpSaL
	1917	Q8NFR9 I17RE_HUMAN	555 80	91	LtqpLLgeqSlL LcqhLLsggSgL
		Q8NGE3 O10P1_HUMAN	9	20	LpefLLlgfSdL
		Q8TCV5 WFDC5_HUMAN	8	19	LlgaLLavgSqL
		Q8TDL5 LPLC1_HUMAN	165	176	LriqLLhklSfL
65		Q8TE82 S3TC1_HUMAN	1025	1036	LegqLLetiSqL
		OSTEOSIPIGO HUMAN	857	868	LegqELensqL Lvfl LflaSfl

1923 Q8TEQ8|PIGO\_HUMAN

857

868 LvflLLflqSfL

### Example 7

### Identification of Motifs within the Somatotropin Derived Peptides

By performing multiple sequence alignment to the sequences of the predicted peptides we can investigate the conservation of specific motifs that are common in most of 25 the sequences. Multiple sequence alignment is performed using the ClustalW algorithm. In order to identify a more robust motif within the peptide sequences, in the case of the somatotropin derived peptides, the lowest similarity hits can be excluded to identify the common amino acids. This process identifies the somatotropin common motif: L-X(3)-L-L-X(3)-S-X-L (SEQ ID NO: 2289) (FIG. 11).

In order to identify the existence of this motif in other protein sequences in the human proteome, the ScanProsite tool was used to search the Prosite database at the Swiss Institute of Bioinformatics. Using the aforementioned motif as a query this motif was identified in 139 locations of 139 different proteins listed in Table 8 (SEQ ID Nos: 1834-1972).

TABLE 8

Amino acid sequences of peptides that contain the somatotropin motif. Somatotropins

Motif: L-X(3)-L-L-X(3)-S-X-L (SEQ ID NO: 2289) Number of Locations: 139 Number of Different Proteins: 139

Accession Number Protein Name	First Ami- no acid	Last Amino acid	Sequence	
O14569 C56D2_HUMAN	164	175	LvgyLLgsaSlL	50
O15287 FANCG_HUMAN	416	427	LceeLLsrtSsL	
O15482 TEX28_HUMAN	338	349	LatvLLvfvStL	
O43914 TYOBP_HUMAN	11	22	LllpLLlavSgL	
O60609 GFRA3_HUMAN	15	26	LmllLLlppSpL	
O75844 FACE1_HUMAN	279	290	LfdtLLeeySvL	
O95747 OXSR1_HUMAN	90	101	LvmkLLsggSvL	55
P01241 SOMA_HUMAN	102	113	LrisLLliqSwL	
P01242 SOM2_HUMAN	102	113	LrisLLliqSwL	
P01243 CSH_HUMAN	102	113	LrisLLlieSwL	
P02750 A2GL_HUMAN	83	94	LpanLLqgaSkL	
P03891 NU2M_HUMAN	149	160	LnvsLLltlSiL	
P04201 MAS_HUMAN	151	162	LvcaLLwalScL	60
P05783 K1C18_HUMAN	338	349	LngiLLhleSeL	00
P07359 GP1BA_HUMAN	3	14	LlllLLllpSpL	
P09848 LPH_HUMAN	35	46	LtndLLhnlSgL	
P11168 GTR2_HUMAN	136	147	LvgaLLmgfSkL	
P12034 FGF5_HUMAN	3	14	LsflLLlffShL	
P13489 RINI_HUMAN	247	258	LcpgLLhpsSrL	
P14902 I23O_HUMAN	196	207	LlkaLLeiaScL	65
P16278 BGAL_HUMAN	135	146	LpawLLekeSiL	
	Number Protein Name  O14569 C56D2_HUMAN O15287 FANCG_HUMAN O15287 FANCG_HUMAN O15482 TEX28_HUMAN O43914 TYOBP_HUMAN O60609 GFRA3_HUMAN O75844 FACE1_HUMAN O95747 OXSR1_HUMAN P01241 SOMA_HUMAN P01242 SOM2_HUMAN P01243 CSH_HUMAN P01243 CSH_HUMAN P03891 NU2M_HUMAN P04201 MAS_HUMAN P05783 K1C18_HUMAN P05783 K1C18_HUMAN P07359 GP1BA_HUMAN P11168 GTR2_HUMAN P11168 GTR2_HUMAN P113489 RINI_HUMAN P13489 RINI_HUMAN P13489 RINI_HUMAN	Ami- Accession no Number Protein Name acid  O14569 C56D2_HUMAN 164 O15287 FANCG_HUMAN 416 O15287 FANCG_HUMAN 338 O43914 TYOBP_HUMAN 11 O60609 GFRA3_HUMAN 15 O75844 FACE1_HUMAN 279 O95747 OXSR1_HUMAN 90 P01241 SOM2_HUMAN 102 P01242 SOM2_HUMAN 102 P01243 CSH_HUMAN 102 P01243 CSH_HUMAN 102 P01243 CSH_HUMAN 31 P03891 NU2M_HUMAN 149 P04201 MAS_HUMAN 159 P05783 K1C18_HUMAN 38 P07359 GP1BA_HUMAN 38 P07359 GP1BA_HUMAN 37 P11168 GTR2_HUMAN 37 P11168 GTR2_HUMAN 37 P12034 FGF5_HUMAN 37 P13489 RINI_HUMAN 37 P13489 RINI_HUMAN 37 P13489 RINI_HUMAN 37	Accession no NumberiProtein Name no Amino NumberiProtein Name no Nu	Accession Number Protein Name  Ami- Last no Amino Number Protein Name  014569 C56D2_HUMAN

## 92 TABLE 9

Table of the amino acid sequences of the peptides identified to contain the serpin motif.

Serpins Motif: L-X(2)-E-E-X-P (SEQ ID NO: 2290) Number of Locations: 314

<u>ins: 30</u>2

				_		11.12		
Amino acid sequences of peptides that contain the somatotropin motif.  Somatotropins					Table of the amino acid sequences of the to contain the serpin mot			
Motif: L-X(3)-L-L-X(3)-S-X-L (SEQ ID NO: 2289) Number of Locations: 139							oins	
Number of Lo Number of Differe				5		Motif: L-X(2)-E-E-X- Number of L		
				-		Number of Differ		
	First	_					First	
Accession	Ami-	Last				Accession	Amino	
# Number Protein Name	no acid	Amino acid	Sequence	10	#	Number Protein Name	acid	
			o equence	-	1072	O00160IMVO1E IIIIMAN	744	
1924 Q8TEZ7 MPRB_HUMAN	127	138	LlahLLqskSeL			O00160 MYO1F_HUMAN O00507 USP9Y_HUMAN	2474	
1925 Q8WWN8 CEND3_HUMAN	1481	1492	LeeqLLqelSsL			O00625 PIR_HUMAN	134	
1926 Q8WZ84 OR8D1_HUMAN 1927 Q92535 PIGC_HUMAN	43 253	54 264	LgmiLLiavSpL LfalLLmsiScL			O14641 DVL2_HUMAN	20	
1928 Q92538 GBF1_HUMAN	1224	1235	LrilLLmkpSvL	15		O14686 MLL2_HUMAN O14709 ZN197_HUMAN	2819 193	
1929 Q92743 HTRA1_HUMAN	262	273	LpvlLLgrsSeL			O14795 UN13B_HUMAN	1499	
1930 Q92935 EXTL1_HUMAN	19	30	LllvLLggfSlL			O15013 ARHGA_HUMAN	199	
1931 Q93074 MED12_HUMAN	401	412	LqtiLLccpSaL			O15055 PER2_HUMAN	994	
1932 Q96DN6 MBD6_HUMAN 1933 Q96GR4 ZDH12_HUMAN	740 48	751 59	LgasLLgdlSsL LtflLLvlgSlL			O15528 CP27B_HUMAN O15534 PER1_HUMAN	297 987	
1934 Q96HP8 T176A_HUMAN	29	40	LaklLLtccSaL	20		O43390 HNRPR_HUMAN	12	
1935 Q96K12 FACR2_HUMAN	380	391	LmnrLLrtvSmL			O60216 RAD21_HUMAN	504	
1936 Q96KP1 EXOC2_HUMAN	339	350	LldkLLetpStL			O60237 MYPT2_HUMAN	339	
1937 Q96MX0 CKLF3_HUMAN	40	51	LkgrLLlaeSgL			O60346 PHLPP_HUMAN O60779 S19A2_HUMAN	483 259	
1938 Q96Q45 AL2S4_HUMAN	387	398	LvvaLLvglSwL			O60885 BRD4_HUMAN	913	
1939 Q96QZ0 PANX3_HUMAN 1940 Q96RQ9 OXLA_HUMAN	136 269	147 280	LssdLLfiiSeL LpraLLsslSgL	25		O75128 COBL_HUMAN	1064	
1941 Q9BY08 EBPL_HUMAN	178	189	LipgLLlwqSwL			O75420 PERQ1_HUMAN	334	
1942 Q9BZ97 TTY13_HUMAN	30	41	LclmLLlagScL			O75787 RENR_HUMAN O75914 PAK3 HUMAN	116 5	
1943 Q9H1Y0 ATG5_HUMAN	85	96	LlfdLLassSaL			O94933 SLIK3_HUMAN	227	
1944 Q9H254 SPTN4_HUMAN	1422	1433	LdkkLLhmeSqL			O94966 UBP19_HUMAN	1251	
1945 Q9H330 CI005_HUMAN	430 175	441	LgkfLLkvdSkL	30		O94986 CE152_HUMAN	847	
1946 Q9H4I8 SEHL2_HUMAN 1947 Q9HCN3 TMEM8_HUMAN	200	186 211	LlqrLLksnShL LpqtLLshpSyL			O94991 SLIK5_HUMAN O95153 RIMB1_HUMAN	230 915	
1948 Q9NQ34 TMM9B_HUMAN	4	15	LwggLLrlgSlL			O95279 KCNK5_HUMAN	443	
1949 Q9NR09 BIRC6_HUMAN	1400	1411	LlkaLLdnmSfL			O95712 PA24B_HUMAN	772	
1950 Q9NRA0 SPHK2_HUMAN	296	307	LgldLLlncSlL			O95881 TXD12_HUMAN	94	
1951 Q9NRU3 CNNM1_HUMAN	156	167	LgalLLlalSaL	35		O96018 APBA3_HUMAN O96024 B3GT4_HUMAN	116 217	
1952 Q9NTT1 U2D3L_HUMAN 1953 Q9NVH2 INT7_HUMAN	99 623	110 634	LskvLLsicSlL LridLLqafSqL			P04275 VWF_HUMAN	1012	
1954 Q9NVM9 CL011_HUMAN	350	361	LtnfLLngrSvL			P05160 F13B_HUMAN	18	
1955 Q9NZD1 GPC5D_HUMAN	60	71	LptqLLfllSvL			P06858 LIPL_HUMAN	279	
1956 Q9P2E9 RRBP1_HUMAN	1226	1237	LrqlLLesqSqL			P07237 PDIA1_HUMAN P07949 RET_HUMAN	307 1033	
1957 Q9P2G4 K1383_HUMAN	397	408	LlnaLLvelSlL	40		P08519 APOA_HUMAN	3880	
1958 Q9P2V4 LRIT1_HUMAN 1959 Q9UDY8 MALT1_HUMAN	541 33	552 44	LpltLLvccSaL		2010	P09769 FGR_HUMAN	497	
1960 Q9UEW8 STK39_HUMAN	138	149	LrepLLrrlSeL LvmkLLsggSmL			P10745 IRBP_HUMAN	708	
1961 Q9UGN4 CM35H_HUMAN	188	199	LlllLLvgaSlL			P11532 DMD_HUMAN P14317 HCLS1_HUMAN	2255 352	
1962 Q9UHD4 CIDEB_HUMAN	189	200	LghmLLgisStL			P16150 LEUK_HUMAN	369	
1963 Q9UIG8 SO3A1_HUMAN	270	281	LcgaLLffsSlL	45	2015	P17025 ZN182_HUMAN	79	
1964 Q9UPA5 BSN_HUMAN	353	364	LgasLLtqaStL			P17600 SYN1_HUMAN	239	
1965 Q9UPX8 SHAN2_HUMAN 1966 Q9Y239 NOD1_HUMAN	609 318	620 329	LtgrLLdpsSpL LsgkLLkgaSkL			P18583 SON_HUMAN P18583 SON_HUMAN	1149 1160	
1967 Q9Y2I2 NTNG1_HUMAN	526	537	LlttLLgtaSpL			P18583 SON_HUMAN	1171	
1968 Q9Y2U2 KCNK7_HUMAN	92	103	LpsaLLfaaSiL		2020	P19484 TFEB_HUMAN	350	
1969 Q9Y2Y8 PRG3_HUMAN	7	18	LpflLLgtvSaL	50		P21333 FLNA_HUMAN	1034	
1970 Q9Y586 MB212_HUMAN	300	311	LngiLLqliScL			P21802 FGFR2_HUMAN P22001 KCNA3_HUMAN	33 152	
1971 Q9Y5X0 SNX10_HUMAN	106	117	LqnaLLlsdSsL			P31629 ZEP2_HUMAN	772	
1972 Q9Y5X5 NPFF2_HUMAN	379	390	LivaLLfilSwL	_	2025	P34925 RYK_HUMAN	578	
						P36955 PEDF_HUMAN	39	
				55		P40189 IL6RB_HUMAN P42898 MTHR_HUMAN	787 598	
Example 8						P48729 KC1A_HUMAN	266	
Exam	pie o				2030	P51512 MMP16_HUMAN	165	
						P52746 ZN142_HUMAN	750	
Identification of Motifs w	ithin t	he Serr	oin Derived			P53370 NUDT6_HUMAN P53801 PTTG_HUMAN	284 167	
Penti		1		60		P53804 TTC3 HUMAN	2001	

## Peptides

The L-X(2)-E-E-X-P (SEQ ID NO: 2290) motif of serpin derived peptides identified the sequences of peptides shown in FIG. 12. Using the ScanProsite tool 314 hits in 302 different proteins were identified. The hits are shown in Table 9 (SEQ ID Nos: 1973-2286).

			First	Last	
		Accession	Amino	Amino	
10	#	Number Protein Name	acid	acid	Sequence
	1973	O00160 MYO1F_HUMAN	744	751	LglEErPe
		O00507 USP9Y_HUMAN	2474	2481	LcpEEePd
		O00625 PIR_HUMAN	134	141	LksEEiPk
		O14641 DVL2_HUMAN	20	27	LdeEEtPy
15		O14686 MLL2_HUMAN	2819	2826	LgpEErPp
		O14709 ZN197_HUMAN O14795 UN13B_HUMAN	193 1499	200 1506	LsqEEnPr LgnEEgPe
		O15013 ARHGA_HUMAN	199	206	LssEEpPt
		O15055 PER2_HUMAN	994	1001	LqlEEaPe
		O15528 CP27B_HUMAN	297	304	LfrEElPa
20		O15534 PER1_HUMAN	987	994	LqlEElPr
20		O43390 HNRPR_HUMAN	12	19	LkeEEePm
		O60216 RAD21_HUMAN	504	511	LppEEpPn
		O60237 MYPT2_HUMAN O60346 PHLPP_HUMAN	339 483	346 490	LyeEEtPk LeaEEkPl
		O60779 S19A2_HUMAN	259	266	LnmEEpPv
		O60885 BRD4_HUMAN	913	920	LedEEpPa
25		O75128 COBL_HUMAN	1064	1071	LerEEkPs
	1991	O75420 PERQ1_HUMAN	334	341	LeeEEePs
		O75787 RENR_HUMAN	116	123	LfsEEtPv
		O75914 PAK3_HUMAN	5	12	LdnEEkPp
		O94933 SLIK3_HUMAN O94966 UBP19_HUMAN	227 1251	234 1258	LqlEEnPw LeaEEePv
30		O94986 CE152 HUMAN	847	854	LknEEvPv
30		O94991 SLIK5_HUMAN	230	237	LqlEEnPw
		O95153 RIMB1_HUMAN	915	922	LngEEcPp
	1999	O95279 KCNK5_HUMAN	443	450	LagEEsPq
		O95712 PA24B_HUMAN	772	779	LkiEEpPs
		O95881 TXD12_HUMAN	94	101	LedEEePk
35		O96018 APBA3_HUMAN O96024 B3GT4_HUMAN	116 217	123 224	LhcEEcPp
		P04275 VWF_HUMAN	1012	1019	LhsEEvPl LqvEEdPv
		P05160 F13B_HUMAN	18	25	LyaEEkPc
		P06858 LIPL_HUMAN	279	286	LlnEEnPs
	2007	P07237 PDIA1_HUMAN	307	314	LkkEEcPa
40		P07949 RET_HUMAN	1033	1040	LseEEtPl
70		P08519 APOA_HUMAN	3880	3887	LpsEEaPt
		P09769 FGR_HUMAN P10745 IRBP_HUMAN	497 708	504 715	LdpEErPt LvvEEaPp
		P11532 DMD_HUMAN	2255	2262	LlvEElPl
		P14317 HCLS1_HUMAN	352	359	LqvEEePv
		P16150 LEUK_HUMAN	369	376	LkgEEePl
45		P17025 ZN182_HUMAN	79	86	LevEEcPa
		P17600 SYN1_HUMAN	239	246	LgtEEfPl
		P18583 SON_HUMAN	1149	1156	LppEEpPt
		P18583 SON_HUMAN P18583 SON_HUMAN	1160 1171	1167 1178	LppEEpPm LppEEpPe
		P19484 TFEB_HUMAN	350	357	LpsEEgPg
50		P21333 FLNA_HUMAN	1034	1041	LprEEgPy
	2022	P21802 FGFR2_HUMAN	33	40	LepEEpPt
		P22001 KCNA3_HUMAN	152	159	LreEErPl
		P31629 ZEP2_HUMAN	772	779	LvsEEsPs
		P34925 RYK_HUMAN	578	585	LdpEErPk LveEEdPf
		P36955 PEDF_HUMAN P40189 IL6RB HUMAN	39 787	46 794	LdsEErPe
55		P42898 MTHR_HUMAN	598	605	LyeEEsPs
		P48729 KC1A_HUMAN	266	273	LrfEEaPd
		P51512 MMP16_HUMAN	165	172	LtfEEvPy
		P52746 ZN142_HUMAN	750	757	LgaEEnPl
		P53370 NUDT6_HUMAN	284	291	LtvEElPa
60		P53801 PTTG_HUMAN P53804 TTC3_HUMAN	167	174 2008	LfkEEnPy LltEEsPs
		P55285 CADH6_HUMAN	2001 116	123	LdrEEkPv
		P55289 CAD12_HUMAN	117	124	LdrEEkPf
	2037	P56645 PER3_HUMAN	929	936	LlqEEmPr
		P59797 SELV_HUMAN	163	170	LlpEEdPe
c =		Q01826 SATB1_HUMAN	409	416	LrkEEdPk
65		Q04725 TLE2_HUMAN	200	207	LveEErPs
	2041	Q06330 SUH_HUMAN	7	14	LpaEEpPa

### TABLE 9-continued

## 94 TABLE 9-continued

Table of the amino acid sequences of the peptides identified to contain the serpin motif.

Serpins

Motif: L-X(2)-E-E-X-P (SEQ ID NO: 2290)

Number of Locations: 314

Number of Different Proteins: 302

Table of the amino acid sequences of the peptides identified to contain the serpin motif.

Serpins

Motif: L-X(2)-E-E-X-P (SEQ ID NO: 2290)

Number of Locations: 314

Number of Different Proteins: 302

Accession # Number Protein Name	First Amino acid	Last Amino acid	Sequence	10	#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
2042 Q06889 EGR3_HUMAN	24	31	LypEEiPs	_	2111	Q6UX39 AMTN_HUMAN	114	121	LssEElPq
2043 Q07157 ZO1_HUMAN	1155	1162	LrhEEqPa			Q6ZMY3 SPOC1_HUMAN	184		LskEEpPg
2044 Q13072 BAGE1_HUMAN	19	26	LmkEEsPv			Q6ZN11 ZN793_HUMAN	60		LeqEEaPw
2045 Q13087 PDIA2_HUMAN	497	504	LptEEpPe			Q6ZNL6 FGD5_HUMAN	382		LraEEnPm
2046 Q13255 GRM1_HUMAN	995 954	1002	LtaEEtPl	15		Q6ZV29 PLPL7_HUMAN	854 527		LhrEEgPa
2047 Q13315 ATM_HUMAN 2048 Q13439 GOGA4_HUMAN	2092	961 2099	LpgEEyPl LeqEEnPg			Q70CQ4 UBP31_HUMAN Q70SY1 CR3L2_HUMAN	527 153		LpqEEqPl LekEEpPl
2049 Q13596 SNX1_HUMAN	265	272	LekEElPr			Q7L8C5 SYT13_HUMAN	229		LaeEElPt
2050 Q13634 CAD18_HUMAN	446	453	LdrEEtPw			Q7Z3E5 ARMC9_HUMAN	570		LnsEElPd
2051 Q14028 CNGB1_HUMAN	137	144	LmaEEnPp			Q7Z410 TMPS9_HUMAN	691	698	LacEEaPg
2052 Q14126 DSG2_HUMAN	117	124	LdrEEtPf	20	2121	Q86SP6 GP149_HUMAN	217	224	LcsEEpPr
2053 Q14204 DYHC_HUMAN	3973	3980	LwsEEtPa	20		Q86V87 RAI16_HUMAN	496		LdlEEdPy
2054 Q14315 FLNC_HUMAN	1738	1745	LphEEePs			Q86VQ0 CF152_HUMAN	428		LerEEkPe
2055 Q14524 SCN5A_HUMAN 2056 Q14554 PDIA5_HUMAN	46 166	53 173	LpeEEaPr LkkEEkPl			Q86W50 MET10_HUMAN Q86Y13 DZIP3_HUMAN	454 1192		LsqEEnPe LlpEEfPg
2057 Q14562 DHX8_HUMAN	411	418	LskEEfPd			Q86Y27 BAGE5_HUMAN	19		LmkEEsPv
2058 Q14562 DHX8_HUMAN	441	448	LveEEpPf			Q86Y28 BAGE4_HUMAN	19		LmkEEsPv
2059 Q14573 ITPR3_HUMAN	315	322	LaaEEnPs	25	2128	Q86Y29 BAGE3_HUMAN	19	26	LmkEEsPv
2060 Q14674 ESPL1_HUMAN	613	620	LspEEtPa			Q86Y30 BAGE2_HUMAN	19		LmkEEsPv
2061 Q14676 MDC1_HUMAN	145	152	LtvEEtPr			Q8IU99 FA26C_HUMAN	315		LgqEEpPl
2062 Q14684 RRP1B_HUMAN	244	251	LsaEEiPe			Q8IUA0 WFDC8_HUMAN	217		LqdEEcPl
2063 Q15021 CND1_HUMAN 2064 Q15735 PI5PA_HUMAN	1179 189	1186 196	LgvEEePf LasEEqPp			Q8IV63 VRK3_HUMAN Q8IWY9 CDAN1_HUMAN	438 948		LtyEEkPp LlpEEtPa
2065 Q15788 NCOA1_HUMAN	982	989	LimEErPn	30		Q8IXI1 MIRO2_HUMAN	24		LvgEEfPe
2066 Q15878 CAC1E_HUMAN	797	804	LnrEEaPt	50		Q8IXI2 MIRO1_HUMAN	24		LvsEEfPe
2067 Q2TAL6 VWC2_HUMAN	179	186	LctEEgPl			Q8IYS5 OSCAR_HUMAN	122		LvtEElPr
2068 Q32MZ4 LRRF1_HUMAN	82	89	LrvEErPe			Q8IZ26 ZNF34_HUMAN	251		LhtEEkPy
2069 Q32P28 P3H1_HUMAN	215	222	LysEEqPq			Q8IZH2 XRN1_HUMAN	1143		LfdEEfPg
2070 Q3KNS1 PTHD3_HUMAN	96	103	LpeEEtPe			Q8IZP0 ABI1_HUMAN	1507		LleEEiPs
2071 Q3ZCX4 ZN568_HUMAN 2072 Q495W5 FUT11_HUMAN	100 1 <b>44</b>	107 151	LeqEEePw LlhEEsPl	35		Q8N201 INT1_HUMAN Q8N309 LRC43_HUMAN	1587 373		LlqEEePl LlvEEsPe
2072 Q495W3 FCTTI_HOMAN 2073 Q52LD8 RFTN2_HUMAN	123	130	LviEEcPl			Q8N3C0 HELC1_HUMAN	451		LsfEEkPv
2074 Q53GL0 PKHO1_HUMAN	189	196	LiqEEdPs			Q8N3C0 HELC1_HUMAN	1579		LatEEdPk
2075 Q53GL0 PKHO1_HUMAN	289	296	LraEEpPt			Q8N475 FSTL5_HUMAN	786	793	LkaEEwPw
2076 Q53GL7 PAR10_HUMAN	693	700	LeaEEpPd			Q8N4L2 TM55A_HUMAN	132		LisEEqPa
2077 Q53H47 SETMR_HUMAN	499	506	LdqEEaPk	40		Q8N752 KC1AL_HUMAN	266		LrfEEvPd
2078 Q567U6 CCD93_HUMAN 2079 Q580R0 CB027_HUMAN	300 41	307 48	LsaEEsPe LelEEaPe			Q8NC74 CT151_HUMAN	178 701		LrgEEkPa LrmEEtPt
2079 Q380R0/CB027_HUMAN 2080 Q587I9/SFT2C_HUMAN	136	143	LrcEEaPs			Q8NE71 ABCF1_HUMAN Q8NEG5 ZSWM2_HUMAN	43		LlrEEePe
2081 Q5H9T9 CN155_HUMAN	427	434	LlpEEaPr			Q8NEM7 FA48A_HUMAN	115		LdaEElPp
2082 Q5H9T9 CN155_HUMAN	697	704	LpaEEtPi			Q8NEZ4 MLL3_HUMAN	3046		LllEEqPl
2083 Q5H9T9 CN155_HUMAN	736	743	LltEEfPi		2152	Q8NEZ4 MLL3_HUMAN	4023	4030	LvkEEpPe
2084 Q5JUK9 GGED1_HUMAN	38	45	LqqEEpPi	45		Q8NFM7 I17RD_HUMAN	702		LgeEEpPa
2085 Q5JXB2 UE2NL_HUMAN	58	65	LlaEEyPm			Q8NFP4 MDGA1_HUMAN	489		LplEEtPd
2086 Q5MCW4 ZN569_HUMAN 2087 Q5SYB0 FRPD1_HUMAN	60 553	67 560	LeqEEePw LikEEqPp			Q8NHJ6 LIRB4_HUMAN Q8NI51 BORIS_HUMAN	60 120		LdkEEsPa LwlEEgPr
2088 Q5THJ4 VP13D_HUMAN	2943	2950	LtgEEiPf			Q8TBH0 ARRD2_HUMAN	387		LysEEdPn
2089 Q5VYS4 CM033_HUMAN	293	300	LesEEtPn			Q8TDX9 PK1L1_HUMAN	1101		LsaEEsPg
2090 Q5VZP5 DUS27_HUMAN	942	949	LrtEEkPp	50		Q8TE68 ES8L1_HUMAN	408	415	LspEEgPp
2091 Q5VZY2 PPC1A_HUMAN	247	254	LkkEErPt			Q8TER0 SNED1_HUMAN	1083		LrgEEhPt
2092 Q63HR2 TENC1_HUMAN	564	571	LddEEqPt			Q8WU49 CG033_HUMAN	8		LslEEcPw
2093 Q66K74 MAP1S_HUMAN 2094 Q68CZ1 FTM_HUMAN	777		LgaEEtPp			Q8WUA2 PPIL4_HUMAN Q8WUI4 HDAC7_HUMAN	16 943		LytEErPr LveEEePm
2095 Q68DD2 PA24F_HUMAN	1181 470	477	LpaEEtPv LyqEEnPa			Q8WWN8 CEND3_HUMAN	1456		LgqEErPp
2096 Q6BDS2 URFB1_HUMAN	1304	1311	LedEEiPv	55		Q8WZ42 TITIN_HUMAN	12132		LvvEElPv
2097 Q6DCA0 AMERL_HUMAN	183	190	LtrEElPk	33		Q8WZ42 TITIN_HUMAN	13832		LfvEEiPv
2098 Q6DN90 IQEC1_HUMAN	263	270	LhtEEaPa			Q92538 GBF1_HUMAN	1062		LqrEEtPs
2099 Q6DT37 MRCKG_HUMAN	1264	1271	LvpEElPp			Q92738 US6NL_HUMAN	51		LheEElPd
2100 Q6HA08 ASTL_HUMAN	62 208	69 305	LilEEtPe			Q92765 SFRP3_HUMAN	134		LacEElPv LlcEEdPf
2101 Q6IFS5 HSN2_HUMAN 2102 Q6NUN7 CK063_HUMAN	298 74	305 81	LnqEElPp LdeEEsPr			Q92851 CASPA_HUMAN Q92888 ARHG1_HUMAN	70 390		LlsEEdPf LepEEpPg
2103 Q6P2Q9 PRP8_HUMAN	1852	1859	LpvEEqPk	60		Q93008 USP9X_HUMAN	2466		LcpEEePd
2104 Q6P5W5 S39A4_HUMAN	473	480	LvaEEsPe			Q969V6 MKL1_HUMAN	497		LvkEEgPr
2105 Q6P6B1 CH047_HUMAN	249	256	LgkEEqPq		2174	Q96B01 R51A1_HUMAN	55		LrkEEiPv
2106 Q6PD74 P34_HUMAN	141	148	LspEElPe			Q96D15 RCN3_HUMAN	192		LhpEEfPh
2107 Q6PI48 SYDM_HUMAN	488	495	LpkEEnPr			Q96DC7 TMCO6_HUMAN	219		LqaEEaPe
2108 Q6PJ61 FBX46_HUMAN 2109 Q6S8J7 POTE8_HUMAN	246 307	253 314	LrkEErPg LtsEEePq	65		Q96FT7 ACCN4_HUMAN Q96G97 BSCL2_HUMAN	90 326		LslEEqPl LseEEkPd
2110 Q6SZW1 SARM1_HUMAN	307 396	403	LiseEerq LlgEEvPr	33		Q96GW7 PGCB_HUMAN	320 880		LhpEEdPe
2110 QUEEN TIDAKUNI_IIOWAIN	590	703	1.18 بانتهاب		2117	5200 " UL OCD TIOMAIN	000	00/	EmpErcure

TABLE 9-continued

## 96 TABLE 9-continued Table of the amino acid sequences of the peptides identified

Table of the amino acid sequences of the peptides identified to contain the serpin motif. Serpins

Motif: L-X(2)-E-E-X-P (SEO ID NO: 2290) Number of Locations: 314 Number of Different Proteins: 302

Accession

Number|Protein Name

2180 Q96H72|S39AD\_HUMAN

2181 Q96H78|S2544\_HUMAN

2182 Q96J42|TXD15\_HUMAN

2183 Q96JI7|SPTCS\_HUMAN

2184 Q96JL9|ZN333\_HUMAN

2185 Q96JQ0|PCD16\_HUMAN

2186 Q96MZ0|GD1L1\_HUMAN

2187 Q96NZ9|PRAP1\_HUMAN

2188 Q96PQ6|ZN317\_HUMAN

2189 Q96RE7|BTB14\_HUMAN

2190 Q96RG2|PASK\_HUMAN

2192 O96SB3|NEB2 HUMAN

2193 Q96SJ8|TSN18\_HUMAN

2194 Q99102|MUC4\_HUMAN

2195 Q99543|DNJC2\_HUMAN 2196 Q9BQS2|SYT15\_HUMAN

2197 Q9BVI0|PHF20\_HUMAN

2198 Q9BY44|EIF2A\_HUMAN

2199 Q9BY78|RNF26\_HUMAN

2200 Q9BYD3|RM04\_HUMAN

2201 Q9BZA7|PC11X\_HUMAN

2202 Q9BZA8|PC11Y\_HUMAN

2203 Q9C009|FOXQ1\_HUMAN

2204 Q9H095|IQCG\_HUMAN

2206 Q9H2C0|GAN\_HUMAN

2205 Q9H0D2|ZN541\_HUMAN

2207 Q9H2X9|S12A5\_HUMAN

2208 Q9H334|FOXP1\_HUMAN

2209 Q9H3T3|SEM6B\_HUMAN

2210 Q9H579|CT132\_HUMAN

2211 Q9H5V8|CDCP1\_HUMAN

2212 Q9H6F5|CCD86\_HUMAN

2213 Q9H6Z4|RANB3\_HUMAN

2214 Q9H7E9|CH033\_HUMAN

2215 Q9H8Y1|CN115\_HUMAN

2216 Q9H9E1|ANRA2\_HUMAN

2217 Q9H9F9|ARP5\_HUMAN

2218 Q9HAV4|XPO5\_HUMAN

2219 Q9HCE7|SMUF1\_HUMAN

2220 Q9NPR2|SEM4B\_HUMAN

2221 Q9NR50|EI2BG\_HUMAN

2222 Q9NRJ7|PCDBG\_HUMAN

2223 Q9NTN9|SEM4G\_HUMAN

2224 Q9NUR3|CT046\_HUMAN

2225 Q9NVR7|TBCC1\_HUMAN

2226 Q9NX46|ARHL2\_HUMAN

2227 Q9NYB9|ABI2\_HUMAN

2228 Q9P1Y5|K1543\_HUMAN

2229 Q9P1Y5|K1543\_HUMAN

2230 Q9P2E7|PCD10\_HUMAN

2231 Q9P2K9|PTHD2\_HUMAN

2232 O9UBB4|ATX10 HUMAN

2233 Q9UBN6|TR10D\_HUMAN

2234 Q9UBT6|POLK\_HUMAN 2235 Q9UGF5|OR5U1\_HUMAN

2236 Q9UGL1|JAD1B\_HUMAN

2237 Q9UHW9|S12A6\_HUMAN

2238 Q9UIF9|BAZ2A\_HUMAN

2239 O9UIG0|BAZ1B HUMAN

2240 Q9ULD6|PDZD6\_HUMAN

2241 Q9ULG1|INOC1\_HUMAN

2242 Q9ULI4|KI26A\_HUMAN

2243 O9ULO1|TPC1 HUMAN

2244 Q9UMS0|NFU1\_HUMAN

2245 Q9UN72|PCDA7\_HUMAN

2246 Q9UN73|PCDA6\_HUMAN

2247 Q9UN74|PCDA4\_HUMAN

2248 Q9UNA0|ATS5\_HUMAN

2191 Q96RL1|UIMC1\_HUMAN

First

Amino

acid

265

42

80

1940

3106

195

71

109

133

388

435

167

1306

68

36

483

461

356

221

315

347

227

122

149

681

291

26

138

788

227

94

137

13

415

521

364

47

333

200

203

104

138

235

827

938

316

673

289

251

303

879

743

609

75

390

235

1396

29

93

200

200

200

78

36

1196

Last

Amino

acid Sequ

347 LleE

272

1947

3113

202 LdhE

78

116 LegE

140

395

442 LseE

174

75 LqlE 43

490

468

363

228 LthE

322

354

234

129

156 LggE

43

688

298

33

145

795 LatE:

234

101

144

20

422 LfsE

340

210

111

145

242

14

834

945

323

680

296

85

258

310

886

750

616

82

397

242

1403

36

100

207

207

207

488

LcpEEpPv

LdrEEePq

LrtEEtPm

LhsEEgPa

LigEEwPs

LgmEErPy

LleEEiPg

LlaEEtPp

LagEEaPg

LdyEEsPv

LevEEePv

LasEEpPd

LkeEEcPa

LlfEEsPs

LskEElPq

LIsEEtPs

LrlEEgPp

LsaEEiPs

LlkEEfPa

LpaEEvPl

LssEEsPr

LrgEEePr

LgqEElPs

LvtEEtPs

LdrEEtPe

LdrEEaPa

LdrEEaPe

LgpEElPg

45

50

1313

1203

49 LwsE

87

to contain the serpin motif. Serpins Motif: L-X(2)-E-E-X-P (SEQ ID NO: 2290) Number of Locations: 314

Number of Different Proteins: 302

Sequence	10	#	Accession Number Protein Name	First Amino acid	Last Amino acid	Sequence
LleEEdPw			Q9UP95 S12A4_HUMAN	678	685	LrlEEgPp
LmaEEgPw		2250	Q9UPQ7 PZRN3_HUMAN	385	392	LlpEEhPs
LwsEEqPa			Q9UPV0 CE164_HUMAN	488	495	LatEEePp
LleEEaPd			Q9UPW6 SATB2_HUMAN	398	405	LrkEEdPr
LkpEElPs	15		Q9UPW8 UN13A_HUMAN	332	339	LeeEElPe
LyrEEgPp			Q9UPX6 K1024_HUMAN	371	378	LntEEvPd
LdhEEePq			Q9UQ05 KCNH4_HUMAN	761	768	LlgEElPp
LttEEkPr			Q9UQ26 RIMS2_HUMAN	201	208	LrnEEaPq
LeqEEePr			Q9UQ26 RIMS2_HUMAN	1327	1334	LsfEEsPq
LhaEEaPs			Q9Y250 LZTS1_HUMAN	293	300	LayEErPr
LvfEEnPf	20		Q9Y2I6 NLP_HUMAN	759	766	LelEEpPq
LllEEePt	20		Q9Y2K7 JHD1A_HUMAN	661	668	LlnEElPn
LseEEdPa			Q9Y2L6 FRM4B_HUMAN	438	445	LpsEEdPa
LdsEEvPe			Q9Y2V3 RX_HUMAN	126	133	LseEEqPk
LhrEErPn			Q9Y343 SNX24_HUMAN	87	94	LenEElPk
LqlEEfPm			Q9Y3I0 CV028_HUMAN	466	473	LvmEEaPe
LtyEElPg			Q9Y3L3 3BP1_HUMAN	130	137	LseEElPa
LepEEsPg	25		Q9Y3L3 3BP1_HUMAN	494	501	LasEElPs
LheEEpPq			Q9Y3R5 DOP2_HUMAN	1084	1091	LseEElPy
LneEEpPg			Q9Y426 CU025_HUMAN	98	105	LsfEEdPr
LthEEmPq			Q9Y566 SHAN1_HUMAN	1838	1845	LpwEEgPg
LdrEEtPn			Q9Y572 RIPK3_HUMAN	352	359	LnlEEpPs
LdrEEtPn			Q9Y5E2 PCDB7_HUMAN	200	207	LdrEEiPe
LrpEEaPg	30		Q9Y5E3 PCDB6_HUMAN	199	206	LdrEEqPq
LitEEgPn			Q9Y5E4 PCDB5_HUMAN	200	207	LdrEErPe
LggEEpPg			Q9Y5E5 PCDB4_HUMAN	199	206	LdrEEqPe
LdgEEiPv			Q9Y5E6 PCDB3_HUMAN	200	207	LdrEEqPe
LrlEEgPp		2276	Q9Y5E7 PCDB2_HUMAN	202	209	LdrEEqPe
LshEEhPh		2277	Q9Y5F1 PCDBC_HUMAN	200	207	LdyEErPe
LfpEEpPp	35	2278	Q9Y5F2 PCDBB_HUMAN	200	207	LdyEElPe
LvqEErPh		2279	Q9Y5F3 PCDB1_HUMAN	200	207	LdrEEqPe
LatEEpPp		2280	Q9Y5G1 PCDGF_HUMAN	200	207	LdrEEqPh
LnkEElPv			Q9Y5G2 PCDGE_HUMAN	410	417	LdrEEiPe
LanEEkPa			Q9Y5H5 PCDA9_HUMAN	200	207	LdrEEtPe
LapEEvPl			Q9Y5I2 PCDAA_HUMAN	199	206	LdrEEnPq
LcsEEsPe	40		Q9Y5I3 PCDA1_HUMAN	200	207	LdrEEtPe
LivEEcPs	40		Q9Y5Q9 TF3C3_HUMAN	42	49	LsaEEnPd
LfsEEtPg			Q9Y5R2 MMP24_HUMAN	201	208	LtfEEvPy
LnrEEiPv		2200	Q9 I 3K2 WIMF24_HUMAN	201	208	Lucevry
LedEElPa						
LgsEErPf						

### Example 9

A Novel Peptide Derived from the Alpha6 Fibril of Type 4 Collagen

A peptide similar to the short Tumstatin T3 peptide derived from the alpha3 fibril of type IV collagen was identified. This peptide was derived from the alpha6 fibril of type 4 collagen. 55 Its amino acid sequence is LPRFSTMPFIYCNINEVCHY (SEQ ID NO: 2304) as shown in FIG. 13.

TABLE 10

60	Table containing the amino acid sequence of the peptide predicted similar to Tumstatin/Tum4						
	Protein Name	Peptide Location	Peptide sequence				
65	Collagen type IV, alpha6 fibril		LPRFSTMPFIYCNINEVCHY (SEQ ID NO: 2304)				

### Example 10

### Peptide Modifications

One skilled in the art will appreciate that peptides disclosed 5 herein may be modified to increase peptide stability for in vivo administration. To demonstrate the desirability of introducing such modifications, three exemplary peptides were selected where in vivo administration in lung carcinoma xenografts of the naked (unmodified) peptides has shown 10 significant efficacy in suppressing the tumor volume increase.

The three exemplary peptides include a peptide derived from the alpha 5 fibril of type IV collagen, a peptide derived from a TSP1 repeat containing protein properdin, and a peptide derived from a CXC chemokine CXCL1 (FIG. 14). The amino acid sequences of mouse and human peptides are shown in FIG. 14. There are minor differences in the amino acid sequences of the mouse and human sequences for TSP1 derived and CXC derived peptide. These differences do not affect the suggested modifications, as the amino acids that 20 may be associated with peptide instability are common in both the mouse and human sequences. The amino acid sequences of the collagen derived peptides are common in both species.

Amino Acid Modifications Controlling Disulfide Bond For- 25 mation

Under oxidizing conditions, the sulfide groups from two cysteines may cross react to form a disulfide bond. If the two cysteines exist in the same molecule, this bond can be formed intra-molecularly producing a hairpin-like tertiary structure 30 in a peptide molecule. If those two cysteines exist either in the same molecule or in two different molecules (one cysteine in the amino acid sequence of the peptide) the disulfide bond formation can cause dimerization or multimerization of the molecules. This can induce possible peptide aggregation, 35 thereby reducing therapeutic efficacy. In addition, albumin contains a free cysteine that can react with the peptides' free cysteines again forming disulfide bonds. These bonds can cause the peptide to non-specifically bind on the albumin's surface. The peptide binding on the albumin's surface can 40 reduce the effective concentration of the circulating peptide.

To promote therapeutic efficacy and reduce the formation of disulfide bonds, cysteines are substituted, for example, by an aminobutyric acid (Abu), serine or alanine. These amino acids have similar physicochemical properties as cysteines, 45 i.e., they include a polar in side chain polarity, neutral in side chain acidity and are largely hydrophobic. However, they are devoid of sulfide groups, which cause them to be non-reactive under oxidizing conditions. Serine and alanine have somewhat different molecular dimensions than cysteine (serine is longer and alanine is shorter). Substitution with these amino acids can cause secondary modifications in the structure of the original peptide. Aminobutyric acid is a favorable modification as it conserves the physicochemical and structural characteristics of the cysteine without the reactive sulfide 55 group.

When two or more cysteines exist per peptide there are two strategies that can be used in order to prevent disulfide bond formation. If the hairpin tertiary structure of the peptide is significant for its activity, the intramolecular disulfide bond 60 formation can be preformed during the solid state synthesis of the molecule if the synthesis is performed under oxidizing conditions. The purification step of the peptide, based on its molecular weight, will eventually obliterate any multimers formed under the oxidizing conditions and can yield a high 65 purity peptide with a hairpin-like tertiary structure. If this structure is not significant or reduces the peptide's activity,

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then the same strategy as in the case of a single cysteine per molecule can be followed. Both of the cysteines can be substituted by aminobutyric acids, serines or alanines.

Amino Acid Modifications Controlling Pegylation Stability

Pegylation involves the conjugation of polyethylene glycol (PEG) to proteins and peptides. Attaching a PEG increases the molecular weight of a molecule, and yield several significant pharmacological advantages over the unmodified form, which include: improved solubility; reduced dosage frequency without diminished efficacy and potentially reduced toxicity; extended circulating life; and enhanced protection from proteolytic degradation.

The presence of methionines in the amino acid sequence of a peptide may induce a low level oxidation reaction at the sulfur containing chain. This can cause the peptide to be unstable in solution or subject to non-specific interactions. The most important potential problem arising from the presence of methionines is the non-specific interactions of these amino acids with PEG chains. These interactions cause binding of the PEG to the methionines, which may present difficulties in purifying Pegylated peptides (i.e., purifying them to greater than 97% which is required by the U.S. Food and Drug Administration for human administration). The most appropriate strategy for minimizing the effect of the methionines on the Pegylation yield is the substitution of the methionines with isoleucines. Isoleucines have many of the same characteristics as methionines, but no cross-reactivity with the PEG chains.

Another amino acid that may interact non-specifically with PEG chains is lysine. This can reduce the yield of the Pegylation reaction. One strategy to minimize nonspecific interactions with lysine is protecting lysine during chemical synthesis. This extra step may increase the cost of Pegylation. A common modification that can be used in order to avoid lysine protection during Pegylation, is substituting arginine for lysine. Arginine has similar characteristics with lysines and does not affect the Pegylation yield.

### Example 11

### Receptor Identification and Peptide Combinations

There is growing evidence that anti-angiogenic peptides exert their effects by binding to receptors on endothelial cells. Tumstatin has two binding sites for αvβ3 integrins (Maeshima et al., (2001) JBiol Chem 276, 31959-31968), although its anti-angiogenic activity has been connected to the site that is located in the amino-terminal of the fragment. Tumstatin has also been shown to interact with α6β1 integrins (Maeshima et al., (2000) J Biol Chem 275, 23745-23750). The major receptor that has been identified for the anti-angiogenic CXC chemokines is CXCR3 (Strieter et al., (2006) Eur J Cancer 42, 768-778). CXCR3 exists in three alternative splice isoforms, CXCR3A, CXCR3B, and CXCR3-alt. The CXC chemokine ligands of CXCR3 inhibit the proliferation and migration of human microvascular endothelial cells in response to a variety of angiogenic factors. Extensive studies on the mechanistic details of the anti-angiogenic activity of thrombospondin 1, the prototype type 1 thrombospondin repeat-containing protein, have implicated CD36, a 88-kDa transmembrane glycoprotein, as the cell-surface receptor that mediates its effects on endothelial cells (Dawson et al., (1997) J Cell Biol 138, 707-717). CD47 and various integrins have also been mechanistically implicated in the effects of thrombospondin 1 on endothelial cells (Gao et al., (1996) J Biol Chem 271, 21-24).

In order to determine whether peptides identified herein share binding partners with previously identified anti-angiogenic peptides, neutralization studies against these receptors were performed. Endothelial cells were pre-incubated with a range of concentrations of neutralizing monoclonal antibodies that target single receptors, and the activity of the peptides in the angiogenesis assay was then compared to that observed in the absence of neutralizing antibody. The results for neutralization studies of the CXC chemokine-derived peptides, the collagen IV-derived peptides, and the TSP1 repeat-containing peptides are presented herein (FIGS. 15A-15C). In each case, a control where the cells were incubated only in the presence of the antibody solutions and without any peptides was carried out. No effect of the antibody alone on the endothelial cells was observed at any concentration.

In order to determine whether CXCR3 is responsible for the binding of the CXC chemokine derived anti-angiogenic peptides, the proliferation experiments were repeated in the presence of different concentrations of a CXCR3-neutralizing antibody. Two concentrations of the antibody were tested. 20 1 and 10 μg/ml, one below and one above the designated  $ED_{50}$ . In most cases, the activity of the peptide was abrogated in the presence of an increasing concentration of the neutralizing antibody against the CXCR3 receptor. Interestingly, in the cases in which the peptide exhibited a biphasic dose 25 response, the monoclonal antibody did not entirely neutralize the activity of the peptide. This suggests that more than one receptor or more than one mechanism is responsible for the activity of these peptides. By performing similar neutralization studies using monoclonal antibodies against all the 30 known CXC receptors, including CXCR1, CXCR2, and CXCR4, none of these receptors appeared to mediate the anti-angiogenic activity of the peptides.

Noting that the effects of tumstatins are primarily attributed to peptides binding to  $\beta 1$  and  $\beta 3$  integrins (Maeshima et 35 al., (2001) J Biol Chem 276, 31959-31968; Maeshima et al., (2001) J Biol Chem 276, 15240-15248), for collagen-derived peptides monoclonal antibodies directed against the β1 and β3 integrins were tested at two antibody concentrations, 1 and 10 ng/ml (FIG. 14). The activity of the highly potent collagen 40 derived peptides was completely abrogated after pre-incubation with either anti-integrin antibody. In the case of the TSP1 repeat-derived peptides, neutralizing CD36, which is the main TSP1 repeat receptor, abolished the peptides' activity. With increasing antibody concentration, increased endothe- 45 lial cell proliferation was observed relative to the control. It is noteworthy that at these two antibody concentrations for which no direct effect on endothelial cells was observed, the antibodies were potent enough to neutralize the peptide activity. In contrast, blocking CD47, the integrin-associated recep- 50 tor, only partially neutralized the peptide activity.

Based on the information obtained from the neutralization experiments, a systematic method to create and test the effectiveness of combinations of individual peptides as potent angiogenesis inhibitors was developed. By using combina- 55 tions of peptides that bind to different receptors, different pathways were targeted to assess whether there was any modulation of the combined activity in our functional assays. In order to evaluate combinations, a sensitive proliferation assay was selected to analyse changes in peptide activity. The 60 tides' potency. use of multiple peptides targeting multiple targets, with different mechanisms or modes of action, creates the possibility for multiple favorable outcomes, including an increased efficacy of the therapeutic effect, the ability to employ a decreased dosage to obtain an analogous or increased level of 65 efficacy (as a strategy to avoid toxicity), as well as a minimization of, or delay in, the development of resistance (Dorrell

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et al., (2007) Proceedings of the National Academy of Sciences of the United States of America 104, 967-972).

Combinations of two peptides were tested from each of the three major protein families, the type IV collagen fibrils, CXC chemokines, and TSP1 repeat-containing proteins. The peptides used in the combination experiments are derived from the alpha5 fibril of type IV collagen (LRRFSTMPFMFC-NINNVCNF (SEQ ID NO: 2312)), from alpha4 fibril of type IV collagen (YCNIHQVCHYAQRNDRSYWL (SEQ ID NO: 2320)), from a CXC protein GRO-α/CXCL1 (NGRKA-CLNPASPIVKKIIEKMLNS (SEQ ID NO: 2305)), from a protein ENA-78/CXCL5 (NGKEICLDPEAP-FLKKVIQKILD (SEQ ID NO: 2311)), from a TSP1 repeatcontaining protein properdin (GPWEPCSVTCSKGTR-TRRR (SEQ ID NO: 2306)), and from a TSP1 repeatcontaining protein THSD6 (WTRCSSSCGRGVSVRSR (SEQ ID NO: 2321)). One peptide from each family was combined at four different concentrations (0.1, 1, 10, and 30 μg/ml), and the efficacy of these combined peptides was evaluated in a proliferation assay. The peptides were applied in series in order to avoid possible interactions between them, and the viability of the cells was then evaluated. Using the information from the dose-response curves, the data was fit to sigmoidal Hill curves (Chou et al., (2006) Pharmacol Rev 58, 621-681). Based on the estimated Hill curves, isobolograms were calculated to obtain the state space of peptide concentrations with equipotent sums of doses. This data was used to generate graphs of equally effective dose pairs (isoboles) with the same level of effectiveness observed for a single peptide application. In addition to the isobolograms the Combination Indexes (C.I.) for different peptide combinations was also calculated (Chou et al., (1984) Adv Enzyme Regul 22, 27-55) to compare the relative efficacy of the various combinations (FIG. 16).

These analyses indicated a significant synergism between CXC chemokines and TSP1 repeat-containing protein-derived peptides. Thus, it is likely that using specific peptide combinations, provides activity levels similar to those obtained when each of the peptides is used alone, but at significantly lower dosages. In the case of combining a CXC derived peptide with a TSP1 derived peptide, dosage was reduced by one order of magnitude while the same level of efficacy was maintained. Furthermore, when applied at higher concentrations, these two peptides in combination yielded a much higher activity than when either one was applied alone. In the case of the combination of collagen IV-derived peptides with either CXC- or TSP1-derived peptides, a synergism was observed only at lower collagen peptide concentrations. At higher concentrations, the collagenderived peptides were antagonized by the CXC and TSP1 repeat-derived peptides.

These studies indicated that the peptides bind to receptors on the endothelial cell surface. Based on the information from the receptor binding, combinatorial strategies were designed targeting multiple receptors. This analysis supports the conclusion that targeting CD36 or CD47, the primary thrombospondin receptors, and CXCR3, the receptor responsible for the anti-angiogenic activity of CXC chemokine-derived peptides, provided for the synergistic amplification of the peptides' potency.

## Example 12

# Anti-Angiogenic Peptides Arrest Tumor Growth

To characterize the functional effects of anti-angiogenic peptides in mouse models, tumor xenografts were generated

in female nude mice using the NCI H82 lung carcinoma cell line. This cell line was chosen because its aggressiveness results in rapid tumor growth. Three peptides, a collagen derived, a CXC chemokine derived and a TSP1 containing protein derived peptide were administered once a day, intra-5 peritoneally (i.p.), at doses 10 and 20 mg/kg/day, in a 200 microliter solution injection as individual agents and as a combination. The CXC protein GRO-α/CXCL1 derived peptide (human sequence: NGRKACLNPASPIVKKIIEK-MLNS (SEQ ID NO: 2445); mouse sequence: NGREACLD- 10 PEAPLVQKIVQKMLKG (SEQ ID NO: 2441)), the TSP1 repeat-containing protein WISP-1 derived peptide (human sequence: GPWEPCSVTCSKGTRTRRR (SEQ ID NO: 2444); mouse sequence: GPWGPCSVTCSKGTQIRQR (SEQ ID NO: 2440)), and the type IV collagen alpha5 fibril 15 derived peptide (human sequence: LRRFSTMPFMFCNIN-NVCNF (SEQ ID NO: 2442) is the same as mouse sequence: LRRFSTMPFMFCNINNVCNF (SEQ ID NO: 2443)). An equivalent volume of PBS was injected as control. The injections were repeated for 12 days. At 10 mg/kg/day (FIG. 18A) 20 and 20 mg/kg/day (FIG. 18B) the peptides suppressed the development of tumors as a monotherapy. Injections of the combination of a TSP1 containing protein derived peptide and a CXC chemokine derived peptide in a rapidly developing tumor (Day 14 after inoculation) completely arrested 25 tumor growth within 3 days (FIG. 18C).

The results described in Example 12 were carried out using the following materials and methods.

Cell Culture

Primary human umbilical vein endothelial cells (HU-VECs) from a single donor were purchased from Cambrex (Walkersville, Md.). The cells were propagated in EGM-2 medium, consisting of a basal cell medium with 2% FBS, growth factors (hbFGF and VEGF) and antibiotics (gentamicin/amphotericin B). The cells were subcultured according to 35 the supplier's instructions: Once the cells had reached subconfluence, they were washed with HEPES buffer solution and trypsinized. The trypsin was then neutralized with trypsin neutralizing solution (TNS; Cambrex, Walkersville, Md.), and the cells were collected and centrifuged at 1500 rpm for 5 minutes. The supernatant was aspirated, and the cells were resuspended in fresh medium. All the cells used were from

In Vitro Cell Viability Assay

passage 3 to passage 6.

To assess the effects of peptides on the proliferation of 45 endothelial cells the viability and metabolic activity of the cells was monitored in the presence of the agent at different concentrations after various periods of time. The colorimetric cell proliferation reagent WST-1 (4-[3-(4-iodophenyl)-2-(4-nitrophenyl)-2H-5-tetrazolio]-1,3-benzene disulphonate) 50 (Roche, Indianapolis, Ind.) was used as the substrate in an assay that measures the metabolic activity of viable cells (Ishiyama et al., (1996) *Biol Pharm Bull* 19, 1518-1520). The assay is based on the reduction of the red tetrazolium salt WST-1 by viable, metabolically active cells to form yellow 55 formazan crystals that are soluble in the cell culture medium.

The cells were cultured as described above and then trypsinized and resuspended in EGM-2 once they had reached 80% confluence. Cell counts were determined using a hemocytometer.

The proliferation assay involved two steps: during the first step, the cells  $(\sim 2\times 10^3/\text{well}$  in a 96-well microplate) were seeded without any extracellular matrix substrate onto the microwells overnight (8 hours). The initial cell culture medium was then removed, and the candidate peptides, dissolved in cell culture medium with growth factors and serum, were added to the wells. The viability of the cells was deter-

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mined after a 3-day exposure to the peptide solution. Each peptide was tested at seven different concentrations: 0.01, 0.1, 1 and 10  $\mu$ g/ml and 20, 30 and 40  $\mu$ g/ml. Each of the concentrations was tested simultaneously in quadruplicate, and each of the experiments was repeated two times. As a positive control (i.e., decreasing viability) 100 ng/ml (0.22 μM) TNP-470 (O-(chloro-acetyl-carbamoyl) fumagillol, a synthetic analogue of fumagillin was applied; 0.46 kDa, provided by NCI) along with the full medium. As a negative control (equivalent to normal viability) the cells were cultured without any test agent in full medium, containing growth factors and serum. The cells were then incubated with the WST-1 reagent for approximately 3 hours. During the incubation period, viable cells convert, in their mitochondria, the red WST-1 to yellow formazan crystals that dissolve in the medium. The second step of the assay involved the quantification of the changes in proliferation by measuring the changes in the color of the metabolized substrate. The samples were read at a wavelength of 570 nm in an ELISA plate reader Victor 3V (Perkin Elmer). The amount of color produced was directly proportional to the number of viable

Monoclonal Antibody Neutralization Assay

In the monoclonal antibody neutralization experiments the endothelial cell proliferation assay was repeated in the presence of varying concentrations of monoclonal antibodies against specific receptors. The endothelial cells were seeded overnight in 96 well plates in full growth factor and serum medium. The medium was removed and replaced with medium containing different monoclonal antibody solutions for beta1 integrins (R&D Systems, MAB17781) alphavbeta3 integrins (R&D Systems, MAB3050), CXCR3 (R&D Systems, MAB1685), CD36 (BD Pharmingen, CB38 (NL07)) and CD47 (BD Pharmingen, B6H12). The cells were incubated for two hours with the antibody solutions. After the two hours the peptide solutions at different concentrations were added in the wells. As a control a set of cells was incubated only in the presence of the monoclonal antibody solutions and without any peptides. The cells were incubated for three days and a cell viability estimation was performed similarly to the proliferation assay.

Isobologram and Combination Index Calculation

The proliferation experiments described above were carried out with peptide combinations. In the combination experiments, the cells were seeded in 96-well microplates using the same cell density as described above, i.e., approximately 2000 cells per well. The cells were allowed to attach overnight (6-8 hours) in full growth factor and serum medium. The full medium was withdrawn and a solution of a single peptide was applied in dose response concentrations of 0.1, 1, 10 and 30  $\mu$ g/ml. These solutions were prepared and applied in growth factor and serum free medium. After two hours the solutions of the first peptide were withdrawn and the solutions of the second peptide were applied in a growth factor and serum free medium. The concentrations at which the second peptide was applied were the same as the concentrations of the first, i.e. in the case that the first peptide was applied at 10 µg/ml, the second was also applied at 10 µg/ml. In addition to the combinations each of the peptides was applied alone for reference. After twenty-four hours the WST-1 dye was applied and the number of live cells was estimated by the optical signal. Dose response sigmoidal curves for a condition "i" were estimated by fitting the data to sigmoidal Hill curves of the type:

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$$E_{i} = E_{i}^{max} \cdot \frac{D_{i}^{n_{i}}}{D_{50,i}^{n_{i}} + D_{i}^{n_{i}}} \rightarrow D_{i} = D_{50,i} \cdot {}^{n_{i}} \sqrt{\frac{E_{i}}{E_{i}^{max} + E_{i}}}$$
 (1)

where E is the effect of the condition "i", in this case the fraction of dead cells,  $E^{max}$  is the maximum observed effect, D is the corresponding dose that yields effectiveness E,  $D_{50}$  is the dose at which half of the maximum effectiveness  $E^{max}$  is observed, and n is the Hill coefficient.

Combining a peptide x with a peptide y and  $D_x^{combo}$  is the applied peptide x concentration in the combination experiment and  $D_y^{combo}$  is the applied peptide y concentration in combination then due to the set up of the experiment, at each experimental condition  $D_x^{combo} = D_y^{combo} = D_c^{combo}$ . In order to construct an isobologram, a graph of equally effective dose pairs (isoboles) for a single peptide effect level (Chou et al., (2006) *Pharmacol Rev* 58, 621-681):

$$\frac{D_x^{combo}}{D_x} + \frac{D_y^{combo}}{D_y} = 1$$
 (2)

In the denominator  $D_x$  is the dose for  $D_x^{combo}$  alone that 25 inhibits the proliferation by effectiveness E and  $D_y$  is the dose for  $D_y^{combo}$  alone that inhibits the proliferation by the same effectiveness E. Also  $D_x^{combo} = D_y^{combo} = D^{combo}$ . Solving equation 2 for a single dose:

$$D_x = \frac{D_y \cdot D^{combo}}{D_y - D^{combo}} \tag{3}$$

After substituting the dose response of the combination  $D^{combo}$  with the corresponding sigmoidal equation 1 as fitted by the experimental data, equation 3 becomes:

$$D_{x} = \frac{D_{y} \cdot D_{50}^{combo} \cdot {}^{n}_{combo} \sqrt{\frac{E_{combo}}{E_{combo}^{max} + E_{combo}}}}{D_{y} - D_{50}^{combo} \cdot {}^{n}_{combo} \sqrt{\frac{E_{combo}}{E_{combo}^{max}} + E_{combo}}}}$$

$$(4)$$

The isobologram is the plot of these concentrations that the effectiveness of an agent alone is the same as the effectiveness of the same agent in combination,  $\mathbf{E}_{combo} = \mathbf{E}_y$  thus equation 4 becomes:

$$D_{x} = \frac{D_{y} \cdot D_{50}^{combo} \cdot {}^{n}combo}{D_{y} - D_{50}^{combo} \cdot {}^{n}combo} \sqrt{\frac{E_{y}}{E_{combo}^{max} + E_{y}}}$$

$$D_{y} - D_{50}^{combo} \cdot {}^{n}combo} \sqrt{\frac{E_{y}}{E_{combo}^{max} + E_{y}}}$$
(5)

But the effectiveness for y alone is defined according to the Hill equation as:

$$E_{y} = E_{y}^{max} \cdot \frac{D_{y}^{n_{y}}}{D_{50, y}^{n_{y}} + D_{y}^{n_{y}}}$$
 (6)

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Thus after substituting equation 6 into 5:

$$D_{x} = D_{y} \cdot D_{50}^{combo} \cdot \frac{E_{y}^{max} \cdot \frac{D_{y}^{ny}}{D_{50,y}^{ny} + D_{y}^{ny}}}{E_{combo}^{max} \cdot E_{y}^{max} \cdot \frac{D_{y}^{ny}}{D_{50,y}^{ny} + D_{y}^{ny}}}$$

$$D_{y} - D_{50}^{combo} \cdot \frac{E_{y}^{max} \cdot \frac{D_{y}^{ny}}{D_{50,y}^{ny} + D_{y}^{ny}}}{E_{combo}^{ny} \cdot E_{combo}^{ny} + E_{y}^{max} \cdot \frac{D_{y}^{ny}}{D_{50,y}^{ny} + D_{y}^{ny}}}$$

$$(7)$$

In order to graph the isobolograms we calculate for each  $D_y$  the corresponding  $D_x$  and plot the  $D_x$  vs.  $D_y$  pairs.

The isobolograms are a special case for the combination index equation as introduced by Chou and Talalay (Chou et al., (1984) *Adv Enzyme Regul* 22, 27-55). The generic equation for the combination index calculation is expressed:

$$CI = \frac{D_x^{combo}}{D_x} + \frac{D_y^{combo}}{D_y} \tag{8}$$

If CI<1 the drug combination effect is synergistic; if C.I.=1 the drug combination effect is additive; whereas if CI>1 the drug combination effect is antagonistic.

In Vivo Tumor Xenograft Models

A population of 10<sup>6</sup> cells were washed twice in PBS and gently resuspended to generate a single cell suspension. The cells were mixed with Matrigel (BD Biosciences) in a final 60% cell solution. Subsequently, the cells were injected into the flank area of immunosuppressed nude mice in a total volume of 100 µl. Following growth incubation of 5 to 6 days, the tumor size volume was calculated by measurements of tumor dimensions with calipers. Tumor growth was monitored to an initial average size of 100 mm<sup>3</sup>, which developed within 6 days after inoculation. Peptides were administered once a day, intraperitoneally (i.p.), in doses of 10 mg/kg and 20 mg/kg. In the case of testing a combination each peptide was injected in a two day cycle of a different peptide per day. Equivalent volume of PBS solution was injected as control. The injections were continued for up to 14 days. A total of six animals per group were used for the experiments per peptide per concentration.

## Other Embodiments

From the foregoing description, it will be apparent that variations and modifications may be made to the invention described herein to adopt it to various usages and conditions. Such embodiments are also within the scope of the following claims

The recitation of a listing of elements in any definition of a variable herein includes definitions of that variable as any single element or combination (or subcombination) of listed elements. The recitation of an embodiment herein includes that embodiment as any single embodiment or in combination with any other embodiments or portions thereof.

with any other embodiments or portions thereof.

The following International Patent Application No. PCT/
US2006/035580, entitled COMPOSITIONS HAVING
ANTIANGIOGENIC ACTIVITY AND USES THEREOF,
which was filed on Sep. 12, 2006 may include related subject
matter, and is hereby incorporated by reference in its entirety.

All patents and publications mentioned in this specification are herein incorporated by reference to the same extent as if each independent patent and publication was specifically and individually indicated to be incorporated by reference. In particular, the sequence of each of the individual NCBI reference numbers listed in Tables 1-10 is hereby incorporated by reference in its entirety.

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<213 > ORGANISM: Homo sapiens
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Trp Thr Lys Cys Ser Ala Thr Cys Gly Gly Gly 1 \phantom{\bigg|}
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<211> LENGTH: 11
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<400> SEQUENCE: 68
Trp Ser Ala Cys Thr Arg Ser Cys Gly Gly Gly
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Trp Cys Cys Cys Cys Phe Pro Cys Cys Arg Gly
<210> SEQ ID NO 70
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<213 > ORGANISM: Homo sapiens
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Trp Ser Ala Cys Asn Val Arg Cys Gly Arg Gly
<210> SEQ ID NO 71
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<213 > ORGANISM: Homo sapiens
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Trp Ala Ser Cys Ser Gln Pro Cys Gly Val Gly
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<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Trp Thr Ser Cys Ser Arg Ser Cys Gly Pro Gly
<210> SEQ ID NO 73
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 73
Trp Ser Gln Cys Ser Val Arg Cys Gly Arg Gly
<210> SEQ ID NO 74
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Trp Gly Glu Cys Ser Ser Glu Cys Gly Ser Gly
<210> SEQ ID NO 75
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Trp Thr Arg Cys Ser Ser Ser Cys Gly Arg Gly
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Trp Met Glu Cys Ser Val Ser Cys Gly Asp Gly
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Trp Thr Ala Cys Ser Arg Ser Cys Gly Gly Gly
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<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 79
Trp Ser Glu Cys Ser Arg Thr Cys Gly Glu Gly
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Trp Gly Pro Cys Ser Gly Ser Cys Gly Gln Gly
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<210> SEQ ID NO 81
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<212> TYPE: PRT
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<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 82
Trp Ser Glu Cys Thr Lys Thr Cys Gly Val Gly 1 \phantom{-} 5 \phantom{-} 10
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<212> TYPE: PRT
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Trp Gly Pro Cys Ser Gly Ser Cys Gly Pro Gly
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Trp Ser Pro Cys Ser Asn Arg Cys Gly Arg Gly
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Trp Ser Glu Cys Ser Arg Thr Cys Gly Gly Gly
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<210> SEQ ID NO 86
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 86
Trp Thr Ala Cys Ser Ser Ser Cys Gly Gly Gly
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<212> TYPE: PRT
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Trp Ser Pro Cys Thr Val Thr Cys Gly Gln Gly
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<210> SEQ ID NO 88
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 88
Trp Ser Met Cys Ser Arg Thr Cys Gly Thr Gly
<210> SEQ ID NO 89
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 89
Trp Glu Gly Cys Ser Val Gln Cys Gly Gly Gly 1 5 10
<210> SEQ ID NO 90
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 90
Trp Ser Pro Cys Ser Ala Thr Cys Glu Lys Gly
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<210> SEQ ID NO 91
<211> LENGTH: 11
<212> TYPE: PRT
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Trp Ser Gln Cys Ser Ala Ser Cys Gly Lys Gly
<210> SEQ ID NO 92
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 92
Trp Ser Thr Cys Ser Ser Thr Cys Gly Lys Gly 1 5 10 ^{\circ}
<210> SEQ ID NO 93
<211> LENGTH: 11
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<400> SEQUENCE: 93
Trp Ser Pro Cys Ser Arg Thr Cys Gly Gly Gly
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<210> SEQ ID NO 94

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<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 94
Trp Ser Ala Cys Ser Arg Thr Cys Gly Gly Gly
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<210> SEQ ID NO 95
<211> LENGTH: 11
<212> TYPE: PRT
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Trp Ala Glu Cys Ser His Thr Cys Gly Lys Gly
<210> SEQ ID NO 96
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 96
<210> SEQ ID NO 97
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 97
Trp Ser Gln Cys Thr Ala Ser Cys Gly Gly Gly
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<210> SEQ ID NO 98
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 98
Trp Gly Pro Cys Ser Ala Ser Cys Gly Ser Gly
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<210> SEQ ID NO 99
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Pro Cys Ser Lys Ser Cys Gly Arg Gly
<210> SEQ ID NO 100
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Pro Cys Ser Arg Thr Cys Ser Ala Gly
<210> SEQ ID NO 101
<211> LENGTH: 11
<212> TYPE: PRT
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<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 101
Trp Glu Asp Cys Asp Ala Thr Cys Gly Gly Gly
<210> SEQ ID NO 102
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 102
Trp Thr Pro Cys Ser Arg Thr Cys Gly Lys Gly
<210> SEQ ID NO 103
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 103
Trp Ser Lys Cys Ser Ile Thr Cys Gly Lys Gly
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<210> SEQ ID NO 104
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 104
Trp Ser Glu Cys Ser Arg Thr Cys Gly Gly Gly
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<210> SEQ ID NO 105
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 105
Trp Ser Thr Cys Ser Lys Ala Cys Ala Gly Gly
<210> SEQ ID NO 106
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 106
Trp Ser Gln Cys Ser Lys Thr Cys Gly Arg Gly
<210> SEQ ID NO 107
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 107
Trp Ser Glu Cys Ser Ala Thr Cys Gly Leu Gly
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<210> SEQ ID NO 108
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 108
Trp Gln Gln Cys Thr Val Thr Cys Gly Gly Gly
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<210> SEQ ID NO 109
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ala Pro Cys Ser Lys Ala Cys Gly Gly Gly
<210> SEQ ID NO 110
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 110
Trp Ser Gln Cys Ser Ala Thr Cys Gly Glu Gly 1 \phantom{-} 5 \phantom{-} 10
<210> SEQ ID NO 111
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 111
Trp Ala Arg Cys Glu Asp Gly Cys Ile Arg Gly
<210> SEQ ID NO 112
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 112
<210> SEQ ID NO 113
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 113
Trp Glu Glu Cys Thr Arg Ser Cys Gly Arg Gly
<210> SEQ ID NO 114
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 114
Trp Gly Thr Cys Ser Glu Ser Cys Gly Lys Gly
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<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 115
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Trp Ser Ala Cys Ser Val Ser Cys Gly Gly Gly
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<210> SEQ ID NO 116
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 116
Trp Gly Thr Cys Ser Arg Thr Cys Asn Gly Gly
<210> SEQ ID NO 117
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 117
Trp Ser Gln Cys Ser Ala Ser Cys Gly Gly Gly
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<210> SEQ ID NO 118
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEOUENCE: 118
Trp Leu Ser Cys Gly Ser Leu Cys Leu Leu Gly
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<210> SEQ ID NO 119
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Trp Gly Arg Cys Thr Gly Asp Cys Gly Pro Gly
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<210> SEQ ID NO 120
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 120
Trp Ser Pro Cys Ser Lys Thr Cys Arg Ser Gly
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<210> SEQ ID NO 121
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 121
Trp Thr Pro Cys Pro Arg Met Cys Gln Ala Gly
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<210> SEQ ID NO 122
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 122
Trp Gly Ser Cys Ser Ser Ser Cys Gly Ile Gly
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<210> SEQ ID NO 123
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 123
Trp Thr Glu Cys Ser Gln Thr Cys Gly His Gly
<210> SEQ ID NO 124
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 124
Trp Ser Thr Cys Glu Leu Thr Cys Ile Asp Gly
<210> SEQ ID NO 125
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 125
Trp Gly Asp Cys Ser Arg Thr Cys Gly Gly Gly
<210> SEQ ID NO 126
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 126
Trp Thr Lys Cys Ser Ala Gln Cys Ala Gly Gly
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<210> SEQ ID NO 127
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Leu Cys Ser Arg Ser Cys Asp Ala Gly
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<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Glu Cys Thr Pro Ser Cys Gly Pro Gly
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<210> SEQ ID NO 129
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 129
Trp Gly Glu Cys Ser Ala Gln Cys Gly Val Gly
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<210> SEQ ID NO 130
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 130
Trp Ser Pro Cys Ser Ile Ser Cys Gly Met Gly
<210> SEQ ID NO 131
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 131
Trp Asp Glu Cys Ser Ala Thr Cys Gly Met Gly
1 5
<210> SEQ ID NO 132
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Asp Cys Ser Val Thr Cys Gly Lys Gly
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<210> SEQ ID NO 133
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 133
Trp Ser Glu Cys Asn Lys Ser Cys Gly Lys Gly 1 5 10 ^{\circ}
<210> SEQ ID NO 134
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 134
Trp Ser Glu Cys Thr Lys Leu Cys Gly Gly Gly
<210> SEQ ID NO 135
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 135
Trp Ser Gln Cys Ser Ala Thr Cys Gly Asp Gly
<210> SEQ ID NO 136
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 136
Trp Ala Leu Cys Ser Thr Ser Cys Gly Ile Gly
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<210> SEQ ID NO 137
<211> LENGTH: 11
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<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 137
Trp Ser Lys Cys Ser Ser Asn Cys Gly Gly Gly
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<210> SEQ ID NO 138
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 138
Trp Ser Ser Cys Ser Arg Asp Cys Glu Leu Gly
<210> SEQ ID NO 139
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 139
Trp Ser Pro Cys Ser Ala Ser Cys Gly Gly Gly 1 5 10 ^{\circ}
<210> SEQ ID NO 140
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 140
<210> SEQ ID NO 141
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 141
Trp Ser Glu Cys Leu Val Thr Cys Gly Lys Gly
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<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Val Gln Cys Ser Val Thr Cys Gly Gln Gly
<210> SEQ ID NO 143
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Trp Thr Pro Cys Ser Ala Thr Cys Gly Lys Gly
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<210> SEQ ID NO 144
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 144
Trp Ser Ser Cys Ser Val Thr Cys Gly Gln Gly
<210> SEQ ID NO 145
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Gly Ala Cys Ser Ser Thr Cys Ala Gly Gly
<210> SEQ ID NO 146
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 146
<210> SEQ ID NO 147
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Trp Ser Ser Cys Ser Val Ser Cys Gly Arg Gly
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<210> SEQ ID NO 148
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Trp Ser Gln Cys Ser Val Ser Cys Gly Arg Gly
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<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Gln Glu Cys Thr Lys Thr Cys Gly Glu Gly
<210> SEQ ID NO 150
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Glu Cys Ser Val Thr Cys Gly Lys Gly
<210> SEQ ID NO 151
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 151
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Trp Gly Ser Cys Ser Val Ser Cys Gly Val Gly
<210> SEQ ID NO 152
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 152
Trp Gly Asp Cys Ser Arg Thr Cys Gly Gly Gly
<210> SEQ ID NO 153
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 153
Trp Gly Glu Cys Ser Lys Ser Cys Glu Leu Gly
<210> SEQ ID NO 154
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 154
Trp Ser Ser Cys Ser Lys Thr Cys Gly Lys Gly
1 5
<210> SEQ ID NO 155
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Trp Ser Ile Cys Ser Arg Ser Cys Gly Met Gly
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<210> SEQ ID NO 156
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Thr Lys Cys Thr Val Thr Cys Gly Arg Gly
<210> SEQ ID NO 157
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Gly Glu Cys Ser Arg Thr Cys Gly Gly Gly
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<211> LENGTH: 11
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<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 158
Trp Ser Glu Cys Ser Ala Thr Cys Ala Gly Gly
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<212> TYPE: PRT
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Trp Gly Gln Cys Ser Arg Ser Cys Gly Gly Gly
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<210> SEQ ID NO 160
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Leu Ala Cys Ser Arg Thr Cys Asp Thr Gly
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<211> LENGTH: 11
<212> TYPE: PRT
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<210> SEQ ID NO 162
<211> LENGTH: 11
<212> TYPE: PRT
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Trp Ser Glu Cys Ser Ser Thr Cys Gly Ala Gly
<210> SEQ ID NO 163
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Trp Ser Glu Cys Ser Lys Thr Cys Gly Ser Gly
<210> SEQ ID NO 164
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 164
Trp Thr Ser Cys Pro Ser Ser Cys Lys Glu Gly
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<210> SEQ ID NO 165
<211> LENGTH: 11
<212> TYPE: PRT
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<400> SEQUENCE: 165
Trp Ser Arg Cys Ser Lys Ser Cys Gly Ser Gly
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<211> LENGTH: 11
<212> TYPE: PRT
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Trp Ser Leu Cys Gln Leu Thr Cys Val Asn Gly
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<210> SEQ ID NO 167
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 167
Gly Lys Thr Thr Cys Leu
<210> SEQ ID NO 168
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 168
Gly Ala Asn Leu Cys Leu
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<210> SEQ ID NO 169
<211> LENGTH: 6
<212> TYPE: PRT
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Gly Glu Ala Gln Cys Leu
<210> SEQ ID NO 170
<211> LENGTH: 6
<212> TYPE: PRT
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<400> SEQUENCE: 170
Gly Ala Thr Thr Cys Leu
<210> SEQ ID NO 171
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 171
Gly Ile Arg Ser Cys Leu
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<210> SEQ ID NO 172
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 172
Gly His Arg Ile Cys Leu
<210> SEQ ID NO 173
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<211> LENGTH: 6
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<400> SEQUENCE: 173
Gly Glu Ala Val Cys Leu
<210> SEQ ID NO 174
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Gly Asp His Pro Cys Leu
<210> SEQ ID NO 175
<211> LENGTH: 6
<212> TYPE: PRT
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Gly Cys Val Cys Cys Leu
<210> SEQ ID NO 177
<211> LENGTH: 6
<212> TYPE: PRT
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Gly Leu His Arg Cys Leu
<210> SEQ ID NO 178
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 178
Gly Leu Val Leu Cys Leu
<210> SEQ ID NO 179
<211> LENGTH: 6
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<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 179
Gly Cys Val Cys Cys Leu
<210> SEQ ID NO 180
<211> LENGTH: 6
<212> TYPE: PRT
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<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 180
Gly Pro Glu Asn Cys Leu
<210> SEQ ID NO 181
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Gly Thr Pro Leu Cys Leu
<210> SEQ ID NO 182
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Gly Thr Ile Tyr Cys Leu
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<210> SEQ ID NO 183
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<400> SEQUENCE: 183
Gly His His Val Cys Leu
<210> SEQ ID NO 184
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<212> TYPE: PRT
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Gly Leu Ile Thr Cys Leu
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<211> LENGTH: 6
<212> TYPE: PRT
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<213 > ORGANISM: Homo sapiens
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Pro Phe Ile Tyr Cys
<210> SEQ ID NO 1796
<211> LENGTH: 5
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Pro Phe Asp Pro Cys
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Pro Phe Ile Leu Cys
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Pro Phe Cys Val Cys
<210> SEQ ID NO 1799
<211> LENGTH: 5
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Pro Phe Ser Asp Cys
<210> SEQ ID NO 1800
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Pro Phe Pro Val Cys
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<210> SEQ ID NO 1802
<211> LENGTH: 5
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1802
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Pro Phe Gly Thr Cys
<210> SEQ ID NO 1803
<211> LENGTH: 5
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1803
Pro Phe Ala Phe Cys
<210> SEQ ID NO 1804
<211> LENGTH: 5
<212> TYPE: PRT
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<400> SEQUENCE: 1804
Pro Phe Arg Pro Cys
<210> SEQ ID NO 1805
<211> LENGTH: 5
<212> TYPE: PRT
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Pro Phe Val Thr Cys
<210> SEQ ID NO 1806
<211> LENGTH: 5
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Pro Phe Arg Val Cys
<210> SEQ ID NO 1807
<211> LENGTH: 5
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Pro Phe Ser His Cys
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<211> LENGTH: 5
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Pro Phe Ser Ser Cys
<210> SEQ ID NO 1809
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1809
Pro Phe Pro Thr Cys
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<211> LENGTH: 5
<212> TYPE: PRT
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<400> SEQUENCE: 1810
Pro Phe Pro Asn Cys
<210> SEQ ID NO 1811
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1811
Pro Phe Gly Leu Cys
1 5
<210> SEQ ID NO 1812
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1812
Pro Phe Cys Arg Cys
<210> SEQ ID NO 1813
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Pro Phe Phe Arg Cys
<210> SEQ ID NO 1814
<211> LENGTH: 5
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<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1814
Pro Phe Phe Arg Cys
1 5
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<211> LENGTH: 5
<212> TYPE: PRT
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Pro Phe Thr Gly Cys
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<210> SEQ ID NO 1816
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<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1816
Pro Phe Pro Ser Cys
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<210> SEQ ID NO 1817
<211> LENGTH: 5
<212> TYPE: PRT
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Pro Phe Tyr Thr Cys
<210> SEQ ID NO 1818
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<212> TYPE: PRT
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<400> SEQUENCE: 1818
Pro Phe Arg Leu Cys
<210> SEQ ID NO 1819
<211> LENGTH: 5
<212> TYPE: PRT
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<400> SEQUENCE: 1819
Pro Phe Glu Thr Cys
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<211> TYPE: PRT
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<400> SEQUENCE: 1820
Pro Phe Ser Gln Cys
<210> SEQ ID NO 1821
<211> LENGTH: 5
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
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Pro Phe Ala Ala Cys
<210> SEQ ID NO 1822
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<212> TYPE: PRT
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Pro Phe Trp Ile Cys
<210> SEQ ID NO 1823
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1823
Pro Phe Ser Arg Cys
<210> SEQ ID NO 1824
<211> LENGTH: 5
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<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1824
Pro Phe Gln Asp Cys
<210> SEQ ID NO 1825
<211> LENGTH: 5
<212> TYPE: PRT
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Pro Phe Gln Leu Cys
<210> SEQ ID NO 1826
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1826
Pro Phe Ala Tyr Cys
<210> SEQ ID NO 1827
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1827
Pro Phe Ser Gln Cys
<210> SEQ ID NO 1828
<211> LENGTH: 5
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1828
Pro Phe Ala Val Cys
<210> SEQ ID NO 1829
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1829
Pro Phe Arg Glu Cys
<210> SEQ ID NO 1830
<211> LENGTH: 5
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1830
Pro Phe Thr Thr Cys
<210> SEQ ID NO 1831
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 1831
Pro Phe Thr Thr Cys
<210> SEQ ID NO 1832
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1832
Pro Phe Thr Thr Cys
<210> SEQ ID NO 1833
<211> LENGTH: 5
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1833
Pro Phe Ala Thr Cys
<210> SEQ ID NO 1834
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1834
Leu Val Gly Tyr Leu Leu Gly Ser Ala Ser Leu Leu 1 \phantom{\bigg|} 10
<210> SEQ ID NO 1835
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1835
Leu Cys Glu Glu Leu Leu Ser Arg Thr Ser Ser Leu
1 5
<210> SEQ ID NO 1836
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1836
Leu Ala Thr Val Leu Leu Val Phe Val Ser Thr Leu
<210> SEQ ID NO 1837
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1837
Leu Leu Leu Pro Leu Leu Leu Ala Val Ser Gly Leu
<210> SEQ ID NO 1838
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1838
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Leu Met Leu Leu Leu Leu Pro Pro Ser Pro Leu
<210> SEQ ID NO 1839
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1839
Leu Phe Asp Thr Leu Leu Glu Glu Tyr Ser Val Leu
<210> SEQ ID NO 1840
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1840
Leu Val Met Lys Leu Leu Ser Gly Gly Ser Val Leu
<210> SEQ ID NO 1841
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1841
Leu Arg Ile Ser Leu Leu Leu Ile Gln Ser Trp Leu
1 5
<210> SEQ ID NO 1842
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1842
Leu Arg Ile Ser Leu Leu Leu Ile Gln Ser Trp Leu
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<210> SEQ ID NO 1843
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1843
Leu Arg Ile Ser Leu Leu Leu Ile Glu Ser Trp Leu
<210> SEQ ID NO 1844
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1844
Leu Pro Ala Asn Leu Leu Gln Gly Ala Ser Lys Leu
<210> SEQ ID NO 1845
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1845
Leu Asn Val Ser Leu Leu Leu Thr Leu Ser Ile Leu
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<210> SEQ ID NO 1846
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1846
Leu Val Cys Ala Leu Leu Trp Ala Leu Ser Cys Leu
1 5
<210> SEQ ID NO 1847
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1847
Leu Asn Gly Ile Leu Leu His Leu Glu Ser Glu Leu 1 $\rm 10^{\circ}
<210> SEQ ID NO 1848
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1848
Leu Leu Leu Leu Leu Leu Leu Pro Ser Pro Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1849
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1849
Leu Thr Asn Asp Leu Leu His Asn Leu Ser Gly Leu
<210> SEQ ID NO 1850
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1850
Leu Val Gly Ala Leu Leu Met Gly Phe Ser Lys Leu
<210> SEQ ID NO 1851
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1851
Leu Ser Phe Leu Leu Leu Phe Phe Ser His Leu
1 5
<210> SEQ ID NO 1852
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1852
Leu Cys Pro Gly Leu Leu His Pro Ser Ser Arg Leu
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<210> SEQ ID NO 1853
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1853
Leu Leu Lys Ala Leu Leu Glu Ile Ala Ser Cys Leu
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<210> SEQ ID NO 1854
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1854
Leu Pro Ala Trp Leu Leu Glu Lys Glu Ser Ile Leu
<210> SEQ ID NO 1855
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1855
Leu Val Arg Asp Leu Leu Glu Val Thr Ser Gly Leu 1 $\rm 10^{\circ}
<210> SEQ ID NO 1856
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1856
Leu Val Arg Gly Leu Leu Ala Lys Lys Ser Lys Leu
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<210> SEQ ID NO 1857
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1857
Leu Ile Leu Gly Leu Leu Leu Cys Phe Ser Val Leu
1 5
<210> SEQ ID NO 1858
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1858
Leu Cys Ser Gly Leu Leu Phe Pro Val Ser Cys Leu
<210> SEQ ID NO 1859
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1859
Leu Ser Ile Leu Leu Leu Ser Cys Ser Val Leu
1
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<210> SEQ ID NO 1860

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<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1860
Leu Ala Ser Leu Leu Leu Ile Cys Lys Ser Ser Leu
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<210> SEQ ID NO 1861
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1861
Leu Leu Ala Ser Leu Leu Ser Pro Gly Ser Val Leu
<210> SEQ ID NO 1862
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1862
Leu Pro Pro Arg Leu Leu Ala Arg Pro Ser Leu Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1863
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1863
Leu Glu Asp Met Leu Leu Thr Thr Leu Ser Gly Leu
1 5
<210> SEQ ID NO 1864
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1864
Leu Leu Glu Tyr Leu Leu Tyr Phe Leu Ser Phe Leu
1 5
<210> SEQ ID NO 1865
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1865
Leu Asn Ser Lys Leu Leu Asp Ile Arg Ser Tyr Leu
<210> SEQ ID NO 1866
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEOUENCE: 1866
Leu Ile Ser Phe Leu Leu Ser Leu Ile Ser Leu Leu
<210> SEQ ID NO 1867
<211> LENGTH: 12
<212> TYPE: PRT
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<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1867
Leu Ile Pro Leu Leu Leu Gln Leu Thr Ser Arg Leu
<210> SEQ ID NO 1868
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1868
Leu Asp Val Gly Leu Leu Ala Asn Leu Ser Ala Leu
<210> SEQ ID NO 1869
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1869
Leu Gln Asp Glu Leu Leu Glu Val Val Ser Glu Leu
1 5
<210> SEQ ID NO 1870
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1870
Leu Ala Ile Val Leu Leu Val Thr Ile Ser Leu Leu
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<210> SEQ ID NO 1871
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1871
Leu Cys Gly Ala Leu Leu Cys Ala Pro Ser Leu Leu
<210> SEQ ID NO 1872
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1872
Leu Val Ile Val Leu Leu Gly Phe Lys Ser Phe Leu
<210> SEQ ID NO 1873
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1873
Leu Gly Ala Ser Leu Leu Ala Ala Ser Ser Leu
1 5
<210> SEQ ID NO 1874
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 1874
Leu Val Ala Gly Leu Leu Leu Trp Ala Ser Leu Leu
1 5
<210> SEQ ID NO 1875
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1875
Leu Asn Gly Ile Leu Leu Gln Leu Ile Ser Cys Leu
1 5
<210> SEQ ID NO 1876
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1876
Leu Leu Leu Leu Leu Ser Ile His Ser Ala Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1877
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1877
Leu Leu Arg Ser Leu Leu Gly Met Leu Ser Asp Leu
<210> SEQ ID NO 1878
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1878
Leu Leu Arg Ser Leu Leu Ser Met Leu Ser Asp Leu
              5
<210> SEQ ID NO 1879
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1879
Leu His Ile Ser Leu Leu Leu Ile Glu Ser Arg Leu
<210> SEQ ID NO 1880
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1880
Leu Leu Val Leu Leu Val Ala Leu Ser Ala Leu
1 5
<210> SEQ ID NO 1881
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1881
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Leu Ile Pro Leu Leu Gln Leu Thr Ser Arg Leu
1 5
<210> SEQ ID NO 1882
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1882
Leu Ile Leu Asn Leu Leu Phe Leu Leu Ser Trp Leu
1 5
<210> SEQ ID NO 1883
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1883
Leu Ala Cys Asp Leu Leu Pro Cys Asn Ser Asp Leu
         5
<210> SEQ ID NO 1884
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEOUENCE: 1884
Leu Ala Thr Asp Leu Leu Ser Thr Trp Ser Val Leu
              5
<210> SEQ ID NO 1885
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1885
Leu Leu Tyr Glu Leu Leu Gln Tyr Glu Ser Ser Leu
1 5
<210> SEQ ID NO 1886
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1886
Leu Asn Arg Ala Leu Leu Met Thr Phe Ser Leu Leu
1 5
<210> SEQ ID NO 1887
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1887
Leu Ile Pro Leu Leu Cln Leu Thr Ser Arg Leu
   5
<210> SEQ ID NO 1888
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1888
Leu Pro Gln Leu Leu Leu Arg Met Ile Ser Ala Leu
1 5
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<210> SEQ ID NO 1889
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1889
Leu Ser Lys Asn Leu Leu Ala Gln Ile Ser Ala Leu
             5
<210> SEQ ID NO 1890
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1890
Leu Ser Gln Asp Leu Leu Glu Asp Asn Ser His Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1891
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1891
Leu Arg Glu Ala Leu Leu Ser Ser Arg Ser His Leu
1 5
<210> SEQ ID NO 1892
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1892
Leu Ile Pro Ala Leu Leu Glu Ser Leu Ser Val Leu
1 5
<210> SEQ ID NO 1893
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1893
Leu Val Ile Val Leu Leu Gly Phe Arg Ser Leu Leu
<210> SEQ ID NO 1894
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1894
Leu Trp Asp Asp Leu Leu Ser Val Leu Ser Ser Leu
1
                                    10
<210> SEQ ID NO 1895
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1895
Leu Val Pro Trp Leu Leu Leu Gly Ala Ser Trp Leu
              5
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<210> SEQ ID NO 1896
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1896
Leu Ala Val Leu Leu Ser Leu Pro Ser Pro Leu
<210> SEQ ID NO 1897
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1897
Leu His Asn Ser Leu Leu Gln Arg Lys Ser Lys Leu
1 5
<210> SEQ ID NO 1898
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1898
Leu Phe Pro Ile Leu Leu Cys Glu Ile Ser Thr Leu
             5
<210> SEQ ID NO 1899
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1899
Leu Phe Gly Thr Leu Leu Tyr Phe Asp Ser Val Leu
<210> SEQ ID NO 1900
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1900
Leu Arg Val Glu Leu Leu Ser Ala Ser Ser Leu Leu
        5
<210> SEQ ID NO 1901
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1901
Leu Arg Ile Ala Leu Leu Tyr Ser His Ser Glu Leu
<210> SEQ ID NO 1902
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1902
Leu Gln Glu Gly Leu Leu Gln Leu Asp Ser Ile Leu
1 5
<210> SEQ ID NO 1903
<211> LENGTH: 12
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<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1903
Leu Val Ile Val Leu Leu Gly Phe Arg Ser Leu Leu
1 5
<210> SEQ ID NO 1904
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1904
Leu Leu Asn Phe Leu Leu Pro Val Phe Ser Pro Leu
<210> SEQ ID NO 1905
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1905
Leu Glu Lys Lys Leu Leu His His Leu Ser Asp Leu
<210> SEQ ID NO 1906
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1906
Leu Leu Asn Ser Leu Leu Asp Ile Val Ser Ser Leu
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<210> SEQ ID NO 1907
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1907
Leu Leu Gln Ser Leu Leu Leu Ser Leu Ser Glu Leu
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<210> SEQ ID NO 1908
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1908
Leu Phe Phe Pro Leu Leu Pro Gln Tyr Ser Lys Leu
<210> SEQ ID NO 1909
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1909
Leu Ala Trp Ser Leu Leu Leu Ser Ser Ala Leu
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<210> SEQ ID NO 1910
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 1910
Leu Glu Ser Asp Leu Leu Ile Glu Glu Ser Val Leu
<210> SEQ ID NO 1911
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1911
Leu Arg Leu Leu Leu Glu Ser Val Ser Gly Leu
<210> SEQ ID NO 1912
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1912
Leu Phe Thr Leu Leu Gln His Arg Ser Gln Leu
<210> SEQ ID NO 1913
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1913
Leu Phe Glu Asp Leu Leu Arg Gln Met Ser Asp Leu
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<210> SEQ ID NO 1914
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1914
Leu Ala Gly Ser Leu Leu Lys Glu Leu Ser Pro Leu
1 5
<210> SEQ ID NO 1915
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1915
Leu Leu Pro Cys Leu Leu Gly Val Gly Ser Trp Leu
<210> SEQ ID NO 1916
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1916
Leu Ser Lys Ser Leu Leu Leu Val Pro Ser Ala Leu
<210> SEQ ID NO 1917
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1917
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Leu Thr Gln Pro Leu Leu Gly Glu Gln Ser Leu Leu
<210> SEQ ID NO 1918
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1918
Leu Cys Gln His Leu Leu Ser Gly Gly Ser Gly Leu
<210> SEQ ID NO 1919
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1919
Leu Pro Glu Phe Leu Leu Leu Gly Phe Ser Asp Leu
<210> SEQ ID NO 1920
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1920
Leu Leu Gly Ala Leu Leu Ala Val Gly Ser Gln Leu
1 5
<210> SEQ ID NO 1921
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1921
Leu Arg Ile Gln Leu Leu His Lys Leu Ser Phe Leu
         5
<210> SEQ ID NO 1922
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1922
Leu Glu Gly Gln Leu Leu Glu Thr Ile Ser Gln Leu
<210> SEQ ID NO 1923
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1923
Leu Val Phe Leu Leu Phe Leu Gln Ser Phe Leu
1 5
<210> SEQ ID NO 1924
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1924
Leu Leu Ala His Leu Leu Gln Ser Lys Ser Glu Leu
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<210> SEQ ID NO 1925
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1925
Leu Glu Glu Gln Leu Leu Gln Glu Leu Ser Ser Leu
1 5
<210> SEQ ID NO 1926
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1926
Leu Gly Met Ile Leu Leu Ile Ala Val Ser Pro Leu
<210> SEQ ID NO 1927
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1927
Leu Phe Ala Leu Leu Leu Met Ser Ile Ser Cys Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1928
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1928
Leu Arg Ile Leu Leu Met Lys Pro Ser Val Leu
<210> SEQ ID NO 1929
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1929
Leu Pro Val Leu Leu Gly Arg Ser Ser Glu Leu
<210> SEQ ID NO 1930
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1930
Leu Leu Val Leu Leu Gly Gly Phe Ser Leu Leu
1 5
<210> SEQ ID NO 1931
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1931
Leu Gln Thr Ile Leu Leu Cys Cys Pro Ser Ala Leu
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<210> SEQ ID NO 1932
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1932
Leu Gly Ala Ser Leu Leu Gly Asp Leu Ser Ser Leu
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<210> SEQ ID NO 1933
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1933
Leu Thr Phe Leu Leu Leu Val Leu Gly Ser Leu Leu
<210> SEQ ID NO 1934
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1934
Leu Ala Lys Leu Leu Leu Thr Cys Cys Ser Ala Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1935
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1935
Leu Met Asn Arg Leu Leu Arg Thr Val Ser Met Leu
                5
<210> SEQ ID NO 1936
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1936
Leu Leu Asp Lys Leu Leu Glu Thr Pro Ser Thr Leu
1 5
<210> SEQ ID NO 1937
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1937
Leu Lys Gly Arg Leu Leu Leu Ala Glu Ser Gly Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1938
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1938
Leu Val Val Ala Leu Leu Val Gly Leu Ser Trp Leu
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<210> SEQ ID NO 1939

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<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1939
Leu Ser Ser Asp Leu Leu Phe Ile Ile Ser Glu Leu
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<210> SEQ ID NO 1940
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1940
Leu Pro Arg Ala Leu Leu Ser Ser Leu Ser Gly Leu
<210> SEQ ID NO 1941
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1941
Leu Ile Pro Gly Leu Leu Leu Trp Gln Ser Trp Leu 1 5 10
<210> SEQ ID NO 1942
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1942
Leu Cys Leu Met Leu Leu Leu Ala Gly Ser Cys Leu
1 5
<210> SEQ ID NO 1943
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1943
Leu Leu Phe Asp Leu Leu Ala Ser Ser Ser Ala Leu
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<210> SEQ ID NO 1944
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1944
Leu Asp Lys Lys Leu Leu His Met Glu Ser Gln Leu
<210> SEQ ID NO 1945
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1945
Leu Gly Lys Phe Leu Leu Lys Val Asp Ser Lys Leu
<210> SEQ ID NO 1946
<211> LENGTH: 12
<212> TYPE: PRT
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<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1946
Leu Leu Gln Arg Leu Leu Lys Ser Asn Ser His Leu
<210> SEQ ID NO 1947
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1947
Leu Pro Gln Thr Leu Leu Ser His Pro Ser Tyr Leu
<210> SEQ ID NO 1948
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1948
Leu Trp Gly Gly Leu Leu Arg Leu Gly Ser Leu Leu 1 \phantom{\bigg|} 10
<210> SEQ ID NO 1949
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1949
Leu Leu Lys Ala Leu Leu Asp Asn Met Ser Phe Leu
               5
<210> SEQ ID NO 1950
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1950
Leu Gly Leu Asp Leu Leu Leu Asn Cys Ser Leu Leu
<210> SEQ ID NO 1951
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1951
Leu Gly Ala Leu Leu Leu Leu Ala Leu Ser Ala Leu
<210> SEQ ID NO 1952
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1952
Leu Ser Lys Val Leu Leu Ser Ile Cys Ser Leu Leu
1 5
<210> SEQ ID NO 1953
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 1953
Leu Arg Ile Asp Leu Leu Gln Ala Phe Ser Gln Leu
1 5
<210> SEQ ID NO 1954
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1954
Leu Thr Asn Phe Leu Leu Asn Gly Arg Ser Val Leu
1 5
<210> SEQ ID NO 1955
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1955
Leu Pro Thr Gln Leu Leu Phe Leu Leu Ser Val Leu 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 1956
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1956
Leu Arg Gln Leu Leu Glu Ser Gln Ser Gln Leu
<210> SEQ ID NO 1957
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1957
Leu Leu Asn Ala Leu Leu Val Glu Leu Ser Leu Leu
              5
<210> SEQ ID NO 1958
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1958
Leu Pro Leu Thr Leu Leu Val Cys Cys Ser Ala Leu
<210> SEQ ID NO 1959
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1959
Leu Arg Glu Pro Leu Leu Arg Arg Leu Ser Glu Leu
1 5
<210> SEQ ID NO 1960
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1960
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Leu Val Met Lys Leu Leu Ser Gly Gly Ser Met Leu
1 5
<210> SEQ ID NO 1961
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1961
Leu Leu Leu Leu Leu Val Gly Ala Ser Leu Leu
1 5
<210> SEQ ID NO 1962
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1962
Leu Gly His Met Leu Leu Gly Ile Ser Ser Thr Leu
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<210> SEQ ID NO 1963
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEOUENCE: 1963
Leu Cys Gly Ala Leu Leu Phe Phe Ser Ser Leu Leu
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<210> SEQ ID NO 1964
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1964
Leu Gly Ala Ser Leu Leu Thr Gln Ala Ser Thr Leu
1 5
<210> SEQ ID NO 1965
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1965
Leu Thr Gly Arg Leu Leu Asp Pro Ser Ser Pro Leu
              5
<210> SEQ ID NO 1966
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1966
Leu Ser Gly Lys Leu Leu Lys Gly Ala Ser Lys Leu
             5
<210> SEQ ID NO 1967
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1967
Leu Leu Thr Thr Leu Leu Gly Thr Ala Ser Pro Leu
1 5
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<210> SEQ ID NO 1968
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1968
Leu Pro Ser Ala Leu Leu Phe Ala Ala Ser Ile Leu
             5
<210> SEQ ID NO 1969
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1969
Leu Pro Phe Leu Leu Gly Thr Val Ser Ala Leu
<210> SEQ ID NO 1970
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1970
Leu Asn Gly Ile Leu Leu Gln Leu Ile Ser Cys Leu
1 5
<210> SEQ ID NO 1971
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1971
Leu Gln Asn Ala Leu Leu Leu Ser Asp Ser Ser Leu
1 5
<210> SEQ ID NO 1972
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1972
Leu Ile Val Ala Leu Leu Phe Ile Leu Ser Trp Leu
             5
<210> SEQ ID NO 1973
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1973
Leu Gly Leu Glu Glu Arg Pro Glu
1
<210> SEQ ID NO 1974
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1974
Leu Cys Pro Glu Glu Glu Pro Asp
     5
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<210> SEQ ID NO 1975
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1975
Leu Lys Ser Glu Glu Ile Pro Lys
<210> SEQ ID NO 1976
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1976
Leu Asp Glu Glu Glu Thr Pro Tyr
1 5
<210> SEQ ID NO 1977
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1977
Leu Gly Pro Glu Glu Arg Pro Pro
              5
<210> SEQ ID NO 1978
<211> LENGTH: 8
<211> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1978
Leu Ser Gln Glu Glu Asn Pro Arg
<210> SEQ ID NO 1979
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1979
Leu Gly Asn Glu Glu Gly Pro Glu
<210> SEQ ID NO 1980
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1980
Leu Ser Ser Glu Glu Pro Pro Thr
<210> SEQ ID NO 1981
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1981
Leu Gln Leu Glu Glu Ala Pro Glu
1 5
<210> SEQ ID NO 1982
<211> LENGTH: 8
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<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1982
Leu Phe Arg Glu Glu Leu Pro Ala
1 5
<210> SEQ ID NO 1983
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1983
Leu Gln Leu Glu Glu Leu Pro Arg
<210> SEQ ID NO 1984
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1984
Leu Lys Glu Glu Glu Pro Met
<210> SEQ ID NO 1985
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1985
Leu Pro Pro Glu Glu Pro Pro Asn
               5
<210> SEQ ID NO 1986
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1986
Leu Tyr Glu Glu Glu Thr Pro Lys
<210> SEQ ID NO 1987
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1987
Leu Glu Ala Glu Glu Lys Pro Leu
<210> SEQ ID NO 1988
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1988
Leu Asn Met Glu Glu Pro Pro Val
                5
<210> SEQ ID NO 1989
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 1989
Leu Glu Asp Glu Glu Pro Pro Ala
<210> SEQ ID NO 1990
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1990
Leu Glu Arg Glu Glu Lys Pro Ser
<210> SEQ ID NO 1991
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1991
Leu Glu Glu Glu Glu Pro Ser
<210> SEQ ID NO 1992
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1992
Leu Phe Ser Glu Glu Thr Pro Val
1 5
<210> SEQ ID NO 1993
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1993
Leu Asp Asn Glu Glu Lys Pro Pro
1 5
<210> SEQ ID NO 1994
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1994
Leu Gln Leu Glu Glu Asn Pro Trp
<210> SEQ ID NO 1995
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1995
Leu Glu Ala Glu Glu Glu Pro Val
<210> SEQ ID NO 1996
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1996
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Leu Lys Asn Glu Glu Val Pro Val
<210> SEQ ID NO 1997
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1997
Leu Gln Leu Glu Glu Asn Pro Trp
<210> SEQ ID NO 1998
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 1998
Leu Asn Gly Glu Glu Cys Pro Pro
<210> SEQ ID NO 1999
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 1999
Leu Ala Gly Glu Glu Ser Pro Gln
1 5
<210> SEQ ID NO 2000
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2000
Leu Lys Ile Glu Glu Pro Pro Ser
               5
<210> SEQ ID NO 2001
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2001
Leu Glu Asp Glu Glu Glu Pro Lys
<210> SEQ ID NO 2002
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2002
Leu His Cys Glu Glu Cys Pro Pro
           5
<210> SEQ ID NO 2003
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2003
Leu His Ser Glu Glu Val Pro Leu
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<210> SEQ ID NO 2004
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2004
Leu Gln Val Glu Glu Asp Pro Val
1 5
<210> SEQ ID NO 2005
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2005
Leu Tyr Ala Glu Glu Lys Pro Cys 1 5
<210> SEQ ID NO 2006
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2006
Leu Leu Asn Glu Glu Asn Pro Ser 1 5
<210> SEQ ID NO 2007
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2007
Leu Lys Lys Glu Glu Cys Pro Ala
<210> SEQ ID NO 2008
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2008
Leu Ser Glu Glu Glu Thr Pro Leu
<210> SEQ ID NO 2009
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2009
Leu Pro Ser Glu Glu Ala Pro Thr
1 5
<210> SEQ ID NO 2010
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2010
Leu Asp Pro Glu Glu Arg Pro Thr
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<210> SEQ ID NO 2011
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2011
Leu Val Val Glu Glu Ala Pro Pro
               5
<210> SEQ ID NO 2012
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2012
Leu Leu Val Glu Glu Leu Pro Leu
<210> SEQ ID NO 2013
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2013
Leu Gln Val Glu Glu Pro Val
<210> SEQ ID NO 2014
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2014
Leu Lys Gly Glu Glu Pro Leu
<210> SEQ ID NO 2015
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2015
Leu Glu Val Glu Glu Cys Pro Ala
<210> SEQ ID NO 2016
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2016
Leu Gly Thr Glu Glu Phe Pro Leu
<210> SEQ ID NO 2017
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2017
Leu Pro Pro Glu Glu Pro Pro Thr
1
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<210> SEQ ID NO 2018

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Leu His Pro Glu Glu Asp Pro Glu
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Leu Pro Ala Glu Glu Val Pro Leu
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Leu Val Thr Glu Glu Thr Pro Ser
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Leu Arg Leu Glu Glu Gly Pro Pro
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Leu Glu Glu Glu Leu Pro Glu
<210> SEQ ID NO 2254
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2254
Leu Asn Thr Glu Glu Val Pro Asp
1
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<210> SEQ ID NO 2255

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<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2255
Leu Leu Gly Glu Glu Leu Pro Pro
<210> SEQ ID NO 2256
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2256
Leu Arg Asn Glu Glu Ala Pro Gln
<210> SEQ ID NO 2257
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2257
Leu Ser Phe Glu Glu Ser Pro Gln
              5
<210> SEQ ID NO 2258
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2258
Leu Ala Tyr Glu Glu Arg Pro Arg
1 5
<210> SEQ ID NO 2259
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2259
Leu Glu Leu Glu Glu Pro Pro Gln
1 5
<210> SEQ ID NO 2260
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2260
Leu Leu Asn Glu Glu Leu Pro Asn
<210> SEQ ID NO 2261
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2261
Leu Pro Ser Glu Glu Asp Pro Ala
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<210> SEQ ID NO 2262
<211> LENGTH: 8
<212> TYPE: PRT
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<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2262
Leu Ser Glu Glu Glu Gln Pro Lys
<210> SEQ ID NO 2263
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2263
Leu Glu Asn Glu Glu Leu Pro Lys
<210> SEQ ID NO 2264
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2264
Leu Val Met Glu Glu Ala Pro Glu
1 5
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<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Leu Ser Glu Glu Glu Leu Pro Ala
<210> SEQ ID NO 2266
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Leu Ala Ser Glu Glu Leu Pro Ser
<210> SEQ ID NO 2267
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2267
Leu Ser Glu Glu Glu Leu Pro Tyr
<210> SEQ ID NO 2268
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2268
Leu Ser Phe Glu Glu Asp Pro Arg
1 5
<210> SEQ ID NO 2269
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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<400> SEQUENCE: 2269
Leu Pro Trp Glu Glu Gly Pro Gly
1 5
<210> SEQ ID NO 2270
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2270
Leu Asn Leu Glu Glu Pro Pro Ser
1 5
<210> SEQ ID NO 2271
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Leu Asp Arg Glu Glu Ile Pro Glu 1 5
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<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Leu Asp Arg Glu Glu Gln Pro Gln
<210> SEQ ID NO 2273
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<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2273
Leu Asp Arg Glu Glu Arg Pro Glu
<210> SEQ ID NO 2274
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Leu Asp Arg Glu Glu Gln Pro Glu
<210> SEQ ID NO 2275
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
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Leu Asp Arg Glu Glu Gln Pro Glu
1 5
<210> SEQ ID NO 2276
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2276
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Leu Asp Arg Glu Glu Gln Pro Glu
1 5
<210> SEQ ID NO 2277
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2277
Leu Asp Tyr Glu Glu Arg Pro Glu
<210> SEQ ID NO 2278
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2278
Leu Asp Tyr Glu Glu Leu Pro Glu
<210> SEQ ID NO 2279
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2279
Leu Asp Arg Glu Glu Gln Pro Glu
             5
<210> SEQ ID NO 2280
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2280
Leu Asp Arg Glu Glu Gln Pro His
1 5
<210> SEQ ID NO 2281
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2281
Leu Asp Arg Glu Glu Ile Pro Glu
1 5
<210> SEQ ID NO 2282
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2282
Leu Asp Arg Glu Glu Thr Pro Glu
             5
<210> SEQ ID NO 2283
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2283
Leu Asp Arg Glu Glu Asn Pro Gln
1 5
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<210> SEQ ID NO 2284
<211> LENGTH: 8
<212> TYPE: PRT
<213> ORGANISM: Homo sapiens
<400> SEQUENCE: 2284
Leu Asp Arg Glu Glu Thr Pro Glu
<210> SEQ ID NO 2285
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2285
Leu Ser Ala Glu Glu Asn Pro Asp
<210> SEQ ID NO 2286
<211> LENGTH: 8
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2286
Leu Thr Phe Glu Glu Val Pro Tyr
<210> SEQ ID NO 2287
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     TSP motif
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (2)..(3)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (5)..(7)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (9)..(10)
<223 > OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2287
Trp Xaa Xaa Cys Xaa Xaa Xaa Cys Xaa Xaa Gly
<210> SEQ ID NO 2288
<211> LENGTH: 7
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     collagen motif
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (3)..(5)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2288
Cys Asn Xaa Xaa Xaa Val Cys
               5
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743 744

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<210> SEQ ID NO 2289
<211> LENGTH: 12
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     somatotropin motif
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (2)..(4)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7)..(9)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11)..(11)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2289
Leu Xaa Xaa Xaa Leu Leu Xaa Xaa Xaa Ser Xaa Leu
              5
<210> SEQ ID NO 2290
<211> LENGTH: 7
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     serpin motif
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (2)..(3)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (6) .. (6)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2290
Leu Xaa Xaa Glu Glu Xaa Pro
1 5
<210> SEQ ID NO 2291
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2291
Leu Leu Arg Ile Ser Leu Leu Leu Ile Glu Ser Trp Leu Glu
               5
<210> SEQ ID NO 2292
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Homo sapiens
<400> SEQUENCE: 2292
Leu Leu Arg Ile Ser Leu Leu Leu Thr Gln Ser Trp Leu Glu
               5
                                    10
<210> SEQ ID NO 2293
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: GH2 peptide
<400> SEQUENCE: 2293
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745 746

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Leu Leu His Ile Ser Leu Leu Leu Ile Gln Ser Trp Leu Glu
1 5
<210> SEQ ID NO 2294
<211> LENGTH: 13
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Chorionic
     somatomammotropin peptide
<400> SEQUENCE: 2294
Leu Leu Arg Leu Leu Leu Ile Glu Ser Trp Leu Glu
<210> SEQ ID NO 2295
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Chorionic
     somatomammotropin hormone-like peptide
<400> SEQUENCE: 2295
Leu Leu His Ile Ser Leu Leu Leu Ile Glu Ser Arg Leu Glu
               5
<210> SEQ ID NO 2296
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Transmembrane protein
     45A peptide
<400> SEQUENCE: 2296
Leu Leu Arg Ser Ser Leu Ile Leu Leu Gln Gly Ser Trp Phe
1 5
<210> SEQ ID NO 2297
<211> LENGTH: 12
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: IL-17 receptor C
     peptide
<400> SEQUENCE: 2297
Arg Leu Arg Leu Leu Thr Leu Gln Ser Trp Leu Leu
<210> SEQ ID NO 2298
<211> LENGTH: 13
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Neuropeptide FF
     receptor 2 peptide
<400> SEQUENCE: 2298
Leu Leu Ile Val Ala Leu Leu Phe Ile Leu Ser Trp Leu
1 5
<210> SEQ ID NO 2299
<211> LENGTH: 13
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
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<223> OTHER INFORMATION: Description of Unknown: Brush border myosin-I
     peptide
<400> SEQUENCE: 2299
Leu Met Arg Lys Ser Gln Ile Leu Ile Ser Ser Trp Phe
1 5
<210> SEQ ID NO 2300
<211> LENGTH: 4
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Motif peptide
<400> SEQUENCE: 2300
Asp Glu Ala His
<210> SEQ ID NO 2301
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: DEAH box polypeptide
     peptide
<400> SEQUENCE: 2301
Glu Ile Glu Leu Val Glu Glu Glu Pro Pro Phe 1 \phantom{\bigg|} 5 \phantom{\bigg|} 10
<210> SEQ ID NO 2302
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Caspase 10 peptide
<400> SEQUENCE: 2302
Ala Glu Asp Leu Leu Ser Glu Glu Asp Pro Phe
               5
<210> SEQ ID NO 2303
<211> LENGTH: 11
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: CKIP-1 peptide
<400> SEQUENCE: 2303
Thr Leu Asp Leu Ile Gln Glu Glu Asp Pro Ser
<210> SEQ ID NO 2304
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Unknown
<220> FEATURE:
<223> OTHER INFORMATION: Description of Unknown: Collagen type IV,
     alpha6 fibril peptide
<400> SEQUENCE: 2304
Leu Pro Arg Phe Ser Thr Met Pro Phe Ile Tyr Cys Asn Ile Asn Glu
Val Cys His Tyr
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<210> SEQ ID NO 2305
<211> LENGTH: 24
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223 > OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2305
Asn Gly Arg Lys Ala Cys Leu Asn Pro Ala Ser Pro Ile Val Lys Lys
       5 10
Ile Ile Glu Lys Met Leu Asn Ser
<210> SEQ ID NO 2306
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2306
Gly Pro Trp Glu Pro Cys Ser Val Thr Cys Ser Lys Gly Thr Arg Thr
                                  10
Arg Arg Arg
<210> SEQ ID NO 2307
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2307
Ser Pro Trp Ser Pro Cys Ser Thr Ser Cys Gly Leu Gly Val Ser Thr
Arg Ile
<210> SEQ ID NO 2308
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2308
Thr Glu Trp Thr Ala Cys Ser Lys Ser Cys Gly Met Gly Phe Ser Thr
Arg Val
<210> SEQ ID NO 2309
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2309
Thr Glu Trp Ser Val Cys Asn Ser Arg Cys Gly Arg Gly Tyr Gln Lys
1 5
                      10
Arg Thr Arg
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<210> SEQ ID NO 2310
<211> LENGTH: 22
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2310
Asn Gly Lys Lys Ala Cys Leu Asn Pro Ala Ser Pro Met Val Gln Lys
Ile Ile Glu Lys Ile Leu
<210> SEQ ID NO 2311
<211> LENGTH: 23
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2311
Asn Gly Lys Glu Ile Cys Leu Asp Pro Glu Ala Pro Phe Leu Lys Lys
                                   10
Val Ile Gln Lys Ile Leu Asp
<210> SEQ ID NO 2312
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2312
Leu Arg Arg Phe Ser Thr Met Pro Phe Met Phe Cys Asn Ile Asn Asn
Val Cys Asn Phe
<210> SEQ ID NO 2313
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2313
Phe Cys Asn Ile Asn Asn Val Cys Asn Phe Ala Ser Arg Asn Asp Tyr
Ser Tyr Trp Leu
<210> SEQ ID NO 2314
<211> LENGTH: 6
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
      6xHis tag
<400> SEQUENCE: 2314
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His His His His His
<210> SEQ ID NO 2315
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (3)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8)..(9)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11) .. (11)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (15)..(15)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2315
Cys Asn Xaa Xaa Xaa Val Cys Xaa Xaa Ala Xaa Arg Asn Asp Xaa Ser
                                    10
Tyr Trp Leu
<210> SEO ID NO 2316
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (2)..(3)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7)..(7)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (10)..(11)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(16)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2316
Leu Xaa Xaa Phe Ser Thr Xaa Pro Phe Xaa Xaa Cys Asn Xaa Xaa Xaa
                                    10
Val Cys
<210> SEQ ID NO 2317
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223 > OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
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<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (5)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8) .. (8)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (10)..(17)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2317
Xaa Xaa Pro Phe Xaa Glu Cys Xaa Gly Xaa Xaa Xaa Xaa Xaa Xaa
Xaa Ala Asn
<210> SEQ ID NO 2318
<211> LENGTH: 16
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223 > OTHER INFORMATION: Description of Artificial Sequence: Synthetic
peptide <220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (5)..(5)
<223> OTHER INFORMATION: Ile or Leu
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8)..(8)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (10)..(10)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11)..(11)
<223> OTHER INFORMATION: Arg or Gly
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (12)..(12)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (13)..(14)
<223> OTHER INFORMATION: Tyr or Phe
<400> SEQUENCE: 2318
Xaa Xaa Pro Phe Xaa Glu Cys Xaa Gly Xaa Xaa Xaa Xaa Ala Asn
<210> SEQ ID NO 2319
<211> LENGTH: 23
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (5)..(5)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7)..(14)
<223> OTHER INFORMATION: Any amino acid
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<221> NAME/KEY: MOD_RES
<222> LOCATION: (16)..(16)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (18)..(22)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2319
Glu Cys Leu Trp Xaa Asp Xaa Xaa Xaa Xaa Xaa Xaa Xaa Gly Xaa
Tyr Xaa Xaa Xaa Xaa Cys
<210> SEQ ID NO 2320
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2320
Tyr Cys Asn Ile His Gln Val Cys His Tyr Ala Gln Arg Asn Asp Arg
                                    10
Ser Tyr Trp Leu
<210> SEQ ID NO 2321
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2321
Trp Thr Arg Cys Ser Ser Ser Cys Gly Arg Gly Val Ser Val Arg Ser
                                    10
Arg
<210> SEQ ID NO 2322
<211> LENGTH: 24
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2322
Asn Gly Arg Glu Ala Cys Leu Asp Pro Glu Ala Pro Leu Val Gln Lys
Ile Val Gln Lys Met Leu Lys Gly
           20
<210> SEQ ID NO 2323
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2323
Gly Pro Trp Gly Pro Cys Ser Val Thr Cys Ser Lys Gly Thr Gln Ile
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10
                                                        15
Arg Gln Arg
<210> SEQ ID NO 2324
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2324
Gly Pro Trp Gly Asp Cys Ser Arg Thr Cys Gly Gly Gly Val Gln Phe
Ser Ser Arg
<210> SEQ ID NO 2325
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2325
Gly Pro Trp Gly Glu Cys Ser Arg Thr Cys Gly Gly Gly Val Gln Phe
1
                                  10
Ser His Arg
<210> SEQ ID NO 2326
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2326
Ser Pro Trp Ser Gln Cys Thr Ala Ser Cys Gly Gly Val Gln Thr
1
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Arg
<210> SEQ ID NO 2327
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
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Ser Lys Trp Ser Glu Cys Ser Arg Thr Cys Gly Gly Gly Val Lys Phe
Gln Glu Arg
<210> SEQ ID NO 2328
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<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223 > OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2328
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Thr Gln Trp Thr Ser Cys Ser Lys Thr Cys Asn Ser Gly Thr Gln Ser
1
                                   10
Arg His Arg
<210> SEQ ID NO 2329
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
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Gly Pro Trp Gly Pro Cys Ser Gly Ser Cys Gly Pro Gly Arg Arg Leu
Arg Arg Arg
<210> SEQ ID NO 2330
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
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     peptide
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Thr Glu Trp Ser Ala Cys Ser Lys Thr Cys Gly Met Gly Ile Ser Thr
Arg Val
<210> SEQ ID NO 2331
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<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2331
Thr Ser Trp Ser Gln Cys Ser Lys Thr Cys Gly Thr Gly Ile Ser Thr
Arg Val
<210> SEQ ID NO 2332
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<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2332
Trp Asp Glu Cys Ser Ala Thr Cys Gly Met Gly Met Lys Lys Arg His
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1
Arg
<210> SEQ ID NO 2333
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2333
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Ser Glu Trp Ser Asp Cys Ser Val Thr Cys Gly Lys Gly Met Arg Thr
Arg Gln Arg
<210> SEQ ID NO 2334
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
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     peptide
<400> SEQUENCE: 2334
Gln Pro Trp Gly Thr Cys Ser Glu Ser Cys Gly Lys Gly Thr Gln Thr
Arg Ala Arg
<210> SEQ ID NO 2335
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEOUENCE: 2335
Ser Ala Trp Arg Ala Cys Ser Val Thr Cys Gly Lys Gly Ile Gln Lys
Arg Ser Arg
<210> SEQ ID NO 2336
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2336
Ala Ser Trp Ser Ala Cys Ser Val Ser Cys Gly Gly Ala Arg Gln
Arg Thr Arg
<210> SEQ ID NO 2337
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<212> TYPE: PRT
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<220> FEATURE:
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     peptide
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Thr Glu Trp Thr Ala Cys Ser Lys Ser Cys Gly Met Gly Phe Ser Thr
                      10
Arg Val
<210> SEQ ID NO 2338
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<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
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<400> SEQUENCE: 2338
Ser Gln Trp Ser Pro Cys Ser Arg Thr Cys Gly Gly Gly Val Ser Phe
1
                                   10
Arg Glu Arg
<210> SEQ ID NO 2339
<211> LENGTH: 18
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2339
Gly Pro Trp Ala Pro Cys Ser Ala Ser Cys Gly Gly Ser Gln Ser
Arg Ser
<210> SEQ ID NO 2340
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
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     peptide
<400> SEQUENCE: 2340
Gly Pro Trp Glu Pro Cys Ser Val Thr Cys Ser Lys Gly Thr Arg Thr
                                 10
Arg Arg Arg
<210> SEQ ID NO 2341
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<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2341
Gly Pro Trp Glu Asp Cys Ser Val Ser Cys Gly Gly Gly Glu Gln Leu
Arg Ser Arg
<210> SEQ ID NO 2342
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2342
Ser Pro Trp Thr Lys Cys Ser Ala Thr Cys Gly Gly Gly His Tyr Met
                                   10
Arg Thr Arg
<210> SEQ ID NO 2343
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<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
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<400> SEQUENCE: 2343
Thr Ser Trp Ser Pro Cys Ser Ala Ser Cys Gly Gly Gly His Tyr Gln
                                   10
Arg Thr Arg
<210> SEQ ID NO 2344
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2344
Gln Pro Trp Ser Gln Cys Ser Ala Thr Cys Gly Asp Gly Val Arg Glu
Arg Arg Arg
<210> SEQ ID NO 2345
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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Ser Pro Trp Ser Pro Cys Ser Gly Asn Cys Ser Thr Gly Lys Gln Gln
                                   10
Arg Thr Arg
<210> SEQ ID NO 2346
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2346
Trp Thr Arg Cys Ser Ser Ser Cys Gly Arg Gly Val Ser Val Arg Ser
               5
                                    10
Arg
<210> SEQ ID NO 2347
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2347
Ser Pro Trp Ser Gln Cys Ser Val Arg Cys Gly Arg Gly Gln Arg Ser
Arg Gln Val Arg
            20
<210> SEQ ID NO 2348
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
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<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2348
Thr Glu Trp Ser Val Cys Asn Ser Arg Cys Gly Arg Gly Tyr Gln Lys
Arg Thr Arg
<210> SEQ ID NO 2349
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2349
Thr Glu Trp Ser Ala Cys Asn Val Arg Cys Gly Arg Gly Trp Gln Lys 1 \phantom{-} 10 \phantom{-} 15
Arg Ser Arg
<210> SEQ ID NO 2350
<211> LENGTH: 18
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2350
Ser Pro Trp Ser Pro Cys Ser Thr Ser Cys Gly Leu Gly Val Ser Thr
                                   10
Arg Ile
<210> SEQ ID NO 2351
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2351
Thr Ala Trp Gly Pro Cys Ser Thr Thr Cys Gly Leu Gly Met Ala Thr
Arg Val
<210> SEQ ID NO 2352
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2352
Thr Lys Trp Thr Pro Cys Ser Arg Thr Cys Gly Met Gly Ile Ser Asn
1
                                    1.0
Arg Val
<210> SEQ ID NO 2353
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
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<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
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<222> LOCATION: (4)..(5)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7)..(9)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11)..(12)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(19)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2353
Xaa Xaa Trp Xaa Xaa Cys Xaa Xaa Cys Xaa Xaa Gly Xaa Xaa Xaa
                                    10
Xaa Xaa Xaa
<210> SEO ID NO 2354
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (4)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7)..(7)
<223> OTHER INFORMATION: Thr, Ser or Asn
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8)..(9)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11)..(12)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(20)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2354
Xaa Xaa Trp Xaa Xaa Cys Xaa Xaa Cys Xaa Xaa Gly Xaa Xaa Xaa
                5
                                    10
Xaa Xaa Xaa Xaa
<210> SEQ ID NO 2355
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
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<222> LOCATION: (4)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
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<222> LOCATION: (8) .. (9)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
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<222> LOCATION: (12)..(12)
<223 > OTHER INFORMATION: Any amino acid
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<222> LOCATION: (14)..(16)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (18)..(20)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2355
Xaa Xaa Trp Xaa Xaa Cys Ser Xaa Xaa Cys Gly Xaa Gly Xaa Xaa Xaa
                                   10
Arg Xaa Xaa Xaa
<210> SEQ ID NO 2356
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2356
Gly Pro Trp Gly Asp Cys Ser Arg Thr Cys Gly Gly Gly Val Gln Tyr
                                   10
Thr Met Arg
<210> SEQ ID NO 2357
<211> LENGTH: 17
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2357
Gly Pro Trp Ser Gln Cys Ser Val Thr Cys Gly Asn Gly Thr Gln Glu
Arg
<210> SEQ ID NO 2358
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2358
Gly Pro Trp Ser Glu Cys Ser Val Thr Cys Gly Glu Gly Thr Glu Val
                        10
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Arg
<210> SEQ ID NO 2359
<211> LENGTH: 14
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2359
Gly Pro Trp Gly Asp Cys Ser Arg Thr Cys Gly Gly Gly Val
<210> SEQ ID NO 2360
<211> LENGTH: 17
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2360
Gly Pro Trp Leu Ala Cys Ser Arg Thr Cys Asp Thr Gly Trp His Thr
                                    10
Arg
<210> SEQ ID NO 2361
<211> LENGTH: 14
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2361
Gln Pro Trp Ser Glu Cys Ser Ala Thr Cys Ala Gly Gly Val
1 5
<210> SEQ ID NO 2362
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2362
Gln Pro Trp Ser Glu Cys Ser Ala Thr Cys Ala Gly Gly Val Gln Arg
Gln
<210> SEQ ID NO 2363
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2363
Gly Pro Trp Gly Gln Cys Ser Gly Pro Cys Gly Gly Gly Val Gln Arg
1
                                   10
Arg
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<210> SEQ ID NO 2364
<211> LENGTH: 14
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2364
Gly Pro Trp Thr Lys Cys Thr Val Thr Cys Gly Arg Gly Val
<210> SEQ ID NO 2365
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2365
Gly Pro Trp Gly Glu Cys Ser Arg Thr Cys Gly Gly Gly Val
<210> SEQ ID NO 2366
<211> LENGTH: 15
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2366
Trp Ser Ser Cys Ser Val Thr Cys Gly Gln Gly Arg Ala Thr Arg
                                    10
<210> SEQ ID NO 2367
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2367
Gly Pro Trp Gly Ala Cys Ser Ser Thr Cys Ala Gly Gly Ser Gln Arg
Arg
<210> SEQ ID NO 2368
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2368
Thr Pro Trp Gly Asp Cys Ser Arg Thr Cys Gly Gly Gly Val Ser Ser
1
                                    1.0
Ser Ser Arg
<210> SEQ ID NO 2369
<211> LENGTH: 15
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
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<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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Trp Asp Leu Cys Ser Thr Ser Cys Gly Gly Gly Phe Gln Lys Arg
                                    10
<210> SEQ ID NO 2370
<211> LENGTH: 14
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2370
Ser Pro Trp Ser His Cys Ser Arg Thr Cys Gly Ala Gly Val
<210> SEQ ID NO 2371
<211> LENGTH: 15
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2371
Trp Met Glu Cys Ser Val Ser Cys Gly Asp Gly Ile Gln Arg Arg
<210> SEQ ID NO 2372
<211> LENGTH: 15
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
      peptide
<400> SEQUENCE: 2372
Trp Ser Gln Cys Ser Ala Thr Cys Gly Glu Gly Ile Gln Gln Arg
<210> SEQ ID NO 2373
<211> LENGTH: 17
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2373
Ser Ala Trp Ser Pro Cys Ser Lys Ser Cys Gly Arg Gly Phe Gln Arg
Arq
<210> SEQ ID NO 2374
<211> LENGTH: 18
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2374
Ser Pro Trp Ser Gln Cys Thr Ala Ser Cys Gly Gly Val Gln Thr
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Arg Ser
<210> SEQ ID NO 2375
<211> LENGTH: 16
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2375
Pro Trp Gln Gln Cys Thr Val Thr Cys Gly Gly Gly Val Gln Thr Arg
<210> SEQ ID NO 2376
<211> LENGTH: 17
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2376
Pro Trp Gln Gln Cys Thr Val Thr Cys Gly Gly Gly Val Gln Thr Arg
                                  10
Ser
<210> SEQ ID NO 2377
<211> LENGTH: 17
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2377
Gly Pro Trp Ser Gln Cys Ser Lys Thr Cys Gly Arg Gly Val Arg Lys
                                   10
Arg
<210> SEQ ID NO 2378
<211> LENGTH: 16
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2378
Trp Ser Lys Cys Ser Ile Thr Cys Gly Lys Gly Met Gln Ser Arg Val
<210> SEQ ID NO 2379
<211> LENGTH: 17
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2379
Asn Ser Trp Asn Glu Cys Ser Val Thr Cys Gly Ser Gly Val Gln Gln
             5
                       10
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Arg

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<210> SEQ ID NO 2380
<211> LENGTH: 18
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2380
Gly Pro Trp Gly Gln Cys Ser Ser Ser Cys Ser Gly Gly Leu Gln His
Arg Ala
<210> SEQ ID NO 2381
<211> LENGTH: 15
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2381
Trp Ser Lys Cys Ser Val Thr Cys Gly Ile Gly Ile Met Lys Arg
<210> SEQ ID NO 2382
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2382
Ser Pro Trp Ser Val Cys Ser Ser Thr Cys Gly Glu Gly Trp Gln Thr
1 5
                       10
Arg Thr Arg
<210> SEQ ID NO 2383
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2383
Ser Pro Trp Ser Val Cys Ser Leu Thr Cys Gly Gln Gly Leu Gln Val
Arg Thr Arg
<210> SEQ ID NO 2384
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2384
Ser Pro Trp Ser Leu Cys Ser Phe Thr Cys Gly Arg Gly Gln Arg Thr
1
                                   10
Arg Thr Arg
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<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2385
Ser Pro Trp Ser Ser Cys Ser Val Thr Cys Gly Val Gly Asn Ile Thr
Arg Ile Arg
<210> SEQ ID NO 2386
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2386
Ser Pro Trp Ser Ala Cys Thr Val Thr Cys Ala Gly Gly Ile Arg Glu
                                    10
Arg Thr Arg
<210> SEQ ID NO 2387
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (4)..(5)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8)..(9)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (12)..(12)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(16)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
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<222> LOCATION: (18) . . (18)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (19) .. (19)
<223> OTHER INFORMATION: Arg or Val
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (20)..(20)
<223> OTHER INFORMATION: Any amino acid
<400> SEOUENCE: 2387
Xaa Xaa Trp Xaa Xaa Cys Ser Xaa Xaa Cys Gly Xaa Gly Xaa Xaa
               5
                                  10
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Arg Xaa Xaa Xaa

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<210> SEQ ID NO 2388
<211> LENGTH: 24
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2388
Asn Gly Lys Gln Val Cys Leu Asp Pro Glu Ala Pro Phe Leu Lys Lys
Val Ile Gln Lys Ile Leu Asp Ser
<210> SEQ ID NO 2389
<211> LENGTH: 22
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
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<400> SEQUENCE: 2389
Asp Gly Arg Lys Ile Cys Leu Asp Pro Asp Ala Pro Arg Ile Lys Lys
Ile Val Gln Lys Lys Leu
            2.0
<210> SEQ ID NO 2390
<211> LENGTH: 23
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2390
Asp Gly Arg Glu Leu Cys Leu Asp Pro Lys Glu Asn Trp Val Gln Arg
1
                                    10
Val Val Glu Lys Phe Leu Lys
<210> SEQ ID NO 2391
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (4)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8)..(8)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (9)..(9)
<223> OTHER INFORMATION: Ser, Arg or Thr
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<220> FEATURE: <221> NAME/KEY: MOD\_RES

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<222> LOCATION: (12)..(12)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(16)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
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<222> LOCATION: (18)..(18)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (19) .. (19)
<223> OTHER INFORMATION: Arg or Val
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (20)..(20)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2391
Arg Xaa Xaa Xaa
<210> SEQ ID NO 2392
<211> LENGTH: 60
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2392
Gln Cys Ile Lys Thr Tyr Ser Lys Pro Phe His Pro Lys Phe Ile Lys
Glu Leu Arg Val Ile Glu Ser Gly Pro His Cys Ala Asn Thr Glu Ile
                              25
Ile Val Lys Leu Ser Asp Gly Arg Glu Leu Cys Leu Asp Pro Lys Glu
Asn Trp Val Gln Arg Val Val Glu Lys Phe Leu Lys
<210> SEQ ID NO 2393
<211> LENGTH: 60
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223 > OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2393
Leu Arg Cys Thr Cys Leu Arg Val Thr Leu Arg Val Asn Pro Lys Thr
Ile Gly Lys Leu Gln Val Phe Pro Ala Gly Pro Gln Cys Ser Lys Val
                             25
Glu Val Val Ala Ser Leu Lys Asn Gly Lys Gln Val Cys Leu Asp Pro
Glu Ala Pro Phe Leu Lys Lys Val Ile Gln Lys Ile
                      55
<210> SEQ ID NO 2394
<211> LENGTH: 56
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
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<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2394
Arg Cys Thr Cys Leu Arg Val Thr Leu Arg Val Asn Pro Lys Thr Ile
Gly Lys Leu Gln Val Phe Pro Ala Gly Pro Gln Cys Ser Lys Val Glu
Val Val Ala Ser Leu Lys Asn Gly Lys Gln Val Cys Leu Asp Pro Glu
Ala Pro Phe Leu Lys Lys Val Ile
<210> SEQ ID NO 2395
<211> LENGTH: 57
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2395
Cys Leu Arg Val Thr Leu Arg Val Asn Pro Lys Thr Ile Gly Lys Leu
Gln Val Phe Pro Ala Gly Pro Gln Cys Ser Lys Val Glu Val Val Ala
                               25
Ser Leu Lys Asn Gly Lys Gln Val Cys Leu Asp Pro Glu Ala Pro Phe
                 40
Leu Lys Lys Val Ile Gln Lys Ile Leu
<210> SEQ ID NO 2396
<211> LENGTH: 56
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    polypeptide
<400> SEQUENCE: 2396
Arg Cys Val Cys Leu Gln Thr Thr Gln Gly Val His Pro Lys Met Ile
Ser Asn Leu Gln Val Phe Ala Ile Gly Pro Gln Cys Ser Lys Val Glu
Val Val Ala Ser Leu Lys Asn Gly Lys Glu Ile Cys Leu Asp Pro Glu
Ala Pro Phe Leu Lys Lys Val Ile
<210> SEQ ID NO 2397
<211> LENGTH: 57
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2397
Cys Leu Gln Thr Thr Gln Gly Val His Pro Lys Met Ile Ser Asn Leu
                                   10
Gln Val Phe Ala Ile Gly Pro Gln Cys Ser Lys Val Glu Val Val Ala
         20
                        25
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Ser Leu Lys Asn Gly Lys Glu Ile Cys Leu Asp Pro Glu Ala Pro Phe
                           40
Leu Lys Lys Val Ile Gln Lys Ile Leu
<210> SEQ ID NO 2398
<211> LENGTH: 58
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2398
Met Cys Ile Lys Thr Thr Ser Gly Ile His Pro Lys Asn Ile Gln Ser
Leu Glu Val Ile Gly Lys Gly Thr His Cys Asn Gln Val Glu Val Ile
Ala Thr Leu Lys Asp Gly Arg Lys Ile Cys Leu Asp Pro Asp Ala Pro _{\mbox{\footnotesize 35}}
Arg Ile Lys Lys Ile Val Gln Lys Lys Leu
<210> SEO ID NO 2399
<211> LENGTH: 56
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2399
Arg Cys Met Cys Ile Lys Thr Thr Ser Gly Ile His Pro Lys Asn Ile
                                    10
Gln Ser Leu Glu Val Ile Gly Lys Gly Thr His Cys Asn Gln Val Glu
                               25
Val Ile Ala Thr Leu Lys Asp Gly Arg Lys Ile Cys Leu Asp Pro Asp
                            40
Ala Pro Arg Ile Lys Lys Ile Val
<210> SEQ ID NO 2400
<211> LENGTH: 58
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2400
Cys Val Lys Thr Thr Ser Gln Val Arg Pro Arg His Ile Thr Ser Leu
                                   10
Glu Val Ile Lys Ala Gly Pro His Cys Pro Thr Ala Gln Leu Ile Ala
Thr Leu Lys Asn Gly Arg Lys Ile Cys Leu Asp Leu Gln Ala Pro Leu
                         40
Tyr Lys Lys Ile Ile Lys Lys Leu Leu Glu
    50
<210> SEQ ID NO 2401
<211> LENGTH: 56
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<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
<400> SEQUENCE: 2401
Arg Cys Gln Cys Leu Gln Thr Leu Gln Gly Ile His Leu Lys Asn Ile
Gln Ser Val Lys Val Lys Ser Pro Gly Pro His Cys Ala Gln Thr Glu
Val Ile Ala Thr Leu Lys Asn Gly Gln Lys Ala Cys Leu Asn Pro Ala
Ser Pro Met Val Lys Lys Ile Ile
<210> SEQ ID NO 2402
<211> LENGTH: 56
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2402
Arg Cys Gln Cys Leu Gln Thr Leu Gln Gly Ile His Leu Lys Asn Ile
                                   10
Gln Ser Val Asn Val Arg Ser Pro Gly Pro His Cys Ala Gln Thr Glu
                              25
Val Ile Ala Thr Leu Lys Asn Gly Lys Lys Ala Cys Leu Asn Pro Ala
                           40
Ser Pro Met Val Gln Lys Ile Ile
   50
<210> SEQ ID NO 2403
<211> LENGTH: 56
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2403
Arg Cys Gln Cys Leu Gln Thr Leu Gln Gly Ile His Pro Lys Asn Ile
Gln Ser Val Asn Val Lys Ser Pro Gly Pro His Cys Ala Gln Thr Glu
Val Ile Ala Thr Leu Lys Asn Gly Arg Lys Ala Cys Leu Asn Pro Ala
Ser Pro Ile Val Lys Lys Ile Ile
<210> SEQ ID NO 2404
<211> LENGTH: 58
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    polypeptide
<400> SEQUENCE: 2404
Gln Cys Leu Gln Thr Leu Gln Gly Ile His Pro Lys Asn Ile Gln Ser
     5
                                10
```

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Val Asn Val Lys Ser Pro Gly Pro His Cys Ala Gln Thr Glu Val Ile
                               25
Ala Thr Leu Lys Asn Gly Arg Lys Ala Cys Leu Asn Pro Ala Ser Pro
Ile Val Lys Lys Ile Ile Glu Lys Met Leu
<210> SEQ ID NO 2405
<211> LENGTH: 58
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2405
Gln Cys Leu Gln Thr Leu Gln Gly Ile His Leu Lys Asn Ile Gln Ser
Val Asn Val Arg Ser Pro Gly Pro His Cys Ala Gln Thr Glu Val Ile
Ala Thr Leu Lys Asn Gly Lys Lys Ala Cys Leu Asn Pro Ala Ser Pro
Met Val Gln Lys Ile Ile Glu Lys Ile Leu
    50
<210> SEQ ID NO 2406
<211> LENGTH: 57
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2406
Arg Cys Thr Cys Ile Ser Ile Ser Asn Gln Pro Val Asn Pro Arg Ser
                                    10
Leu Glu Lys Leu Glu Ile Ile Pro Ala Ser Gln Phe Cys Pro Arg Val
            20
                                25
Glu Ile Ile Ala Thr Met Lys Lys Gly Glu Lys Arg Cys Leu Asn Pro
Glu Ser Lys Ala Ile Lys Asn Leu Leu
<210> SEQ ID NO 2407
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(1)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (4)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7)..(9)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11)..(12)
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<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(20)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2407
Xaa Pro Trp Xaa Xaa Cys Xaa Xaa Cys Xaa Xaa Gly Xaa Xaa Xaa
Xaa Xaa Xaa Xaa
<210> SEQ ID NO 2408
<211> LENGTH: 78
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2408
Asn Glu Arg Ala His Gly Gln Asp Leu Gly Thr Ala Gly Ser Cys Leu
Arg Lys Phe Ser Thr Met Pro Phe Leu Phe Cys Asn Ile Asn Asn Val
Cys Asn Phe Ala Ser Arg Asn Asp Tyr Ser Tyr Trp Leu Ser Thr Pro
                           40
Glu Pro Met Pro Met Ser Met Ala Pro Ile Thr Gly Glu Asn Ile Arg
                     55
Pro Phe Ile Ser Arg Cys Ala Val Cys Glu Ala Pro Ala Met
                   70
<210> SEQ ID NO 2409
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2409
Leu Arg Lys Phe Ser Thr Met Pro Phe Leu Phe Cys Asn Ile Asn Asn
                                   10
Val Cys Asn Phe
<210> SEQ ID NO 2410
<211> LENGTH: 78
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2410
Asn Lys Arg Ala His Gly Gln Asp Leu Gly Thr Ala Gly Ser Cys Leu
                        10
Arg Arg Phe Ser Thr Met Pro Phe Met Phe Cys Asn Ile Asn Asn Val
                               25
Cys Asn Phe Ala Ser Arg Asn Asp Tyr Ser Tyr Trp Leu Ser Thr Pro
                           40
Glu Pro Met Pro Met Ser Met Gln Pro Leu Lys Gly Gln Ser Ile Gln
```

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Pro Phe Ile Ser Arg Cys Ala Val Cys Glu Ala Pro Ala Val
65 70 75
<210> SEQ ID NO 2411
<211> LENGTH: 77
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2411
Gln Glu Lys Ala His Asn Gln Asp Leu Gly Glu Ala Gly Ser Cys Leu
Arg Arg Phe Ser Thr Met Pro Phe Ile Tyr Cys Asn Ile Asn Glu Val
Cys His Tyr Ala Arg Arg Asn Asp Lys Ser Tyr Trp Leu Ser Thr Thr
Ala Pro Ile Pro Met Met Pro Val Ser Gln Thr Gln Ile Pro Gln Tyr
Ile Ser Arg Cys Ser Val Cys Glu Ala Pro Ser Gln Ala
<210> SEQ ID NO 2412
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<400> SEQUENCE: 2412
Tyr Cys Asn Ile Asn Glu Val Cys His Tyr Ala Arg Arg Asn Asp Lys
Ser Tyr Trp Leu
<210> SEQ ID NO 2413
<211> LENGTH: 77
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2413
Gln Glu Lys Ala His Asn Gln Asp Leu Gly Leu Ala Gly Ser Cys Leu
1 5 10 15
Ala Arg Phe Ser Thr Met Pro Phe Leu Tyr Cys Asn Pro Gly Asp Val
Cys Tyr Tyr Ala Ser Arg Asn Asp Lys Ser Tyr Trp Leu Ser Thr Thr
                          40
Ala Pro Leu Pro Met Met Pro Val Ala Glu Asp Glu Ile Lys Arg Tyr
Ile Ser Arg Cys Ser Val Cys Glu Ala Pro Ala Ile Ala
                  70
<210> SEQ ID NO 2414
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
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<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2414
Tyr Cys Asn Pro Gly Asp Val Cys Tyr Tyr Ala Ser Arg Asn Asp Lys
Ser Tyr Trp Leu
<210> SEQ ID NO 2415
<211> LENGTH: 77
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<223 > OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2415
Gln Glu Lys Ala His Asn Gln Asp Leu Gly Leu Ala Gly Ser Cys Leu
                                  10
Pro Val Phe Ser Thr Leu Pro Phe Ala Tyr Cys Asn Ile His Gln Val
                              25
Cys His Tyr Ala Gln Arg Asn Asp Arg Ser Tyr Trp Leu Ala Ser Ala
                           40
Ala Pro Leu Pro Met Met Pro Leu Ser Glu Glu Ala Ile Arg Pro Tyr
                       55
Val Ser Arg Cys Ala Val Cys Glu Ala Pro Ala Gln Ala
                    70
<210> SEQ ID NO 2416
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2416
Leu Pro Val Phe Ser Thr Leu Pro Phe Ala Tyr Cys Asn Ile His Gln
                                    10
Val Cys His Tyr
<210> SEQ ID NO 2417
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1) .. (1)
<223> OTHER INFORMATION: Ser, Gly or Gln
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (4)..(5)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (7) .. (7)
<223> OTHER INFORMATION: Thr or Ser
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8)..(9)
<223 > OTHER INFORMATION: Any amino acid
<220> FEATURE:
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<221> NAME/KEY: MOD_RES
<222> LOCATION: (11) ..(11)
<223> OTHER INFORMATION: Gly or Ser
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (12)..(12)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(16)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (17)..(17)
<223> OTHER INFORMATION: Arg or Ser
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (18)..(20)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2417
Xaa Pro Trp Xaa Xaa Cys Xaa Xaa Cys Xaa Xaa Gly Xaa Xaa Xaa
                                  10
Xaa Xaa Xaa Xaa
          20
<210> SEQ ID NO 2418
<211> LENGTH: 77
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2418
Gln Glu Lys Ala His Asn Gln Asp Leu Gly Phe Ala Gly Ser Cys Leu
1
                       10
Pro Arg Phe Ser Thr Met Pro Phe Ile Tyr Cys Asn Ile Asn Glu Val
                            25
Cys His Tyr Ala Arg Arg Asn Asp Lys Ser Tyr Trp Leu Ser Thr Thr
Ala Pro Ile Pro Met Met Pro Val Ser Gln Thr Gln Ile Pro Gln Tyr
Ile Ser Arg Cys Ser Val Cys Glu Ala Pro Ser Gln Ala
             70
<210> SEQ ID NO 2419
<211> LENGTH: 77
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     polypeptide
<400> SEQUENCE: 2419
Gln Glu Lys Ala His Asn Gln Asp Leu Gly Leu Ala Gly Ser Cys Leu
                                  1.0
Ala Arg Phe Ser Thr Met Pro Phe Leu Tyr Cys Asn Pro Gly Asp Val
                    25
Cys Tyr Tyr Ala Ser Arg Asn Asp Lys Ser Tyr Trp Leu Ser Thr Thr
                          40
Ala Pro Leu Pro Met Met Pro Val Ala Glu Asp Glu Ile Lys Pro Tyr
                      55
Ile Ser Arg Cys Ser Val Cys Glu Ala Pro Ala Ile Ala
           70
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<210> SEQ ID NO 2420
<211> LENGTH: 20
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1) .. (1)
<223> OTHER INFORMATION: Ser, Thr, Gly, Gln or Ala
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (2)..(2)
<223> OTHER INFORMATION: Pro, Glu, Ser, Ala, Gln or Lys
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (4) .. (4)
<223> OTHER INFORMATION: Ser, Thr, Gly, Glu, Asp, Arg or Ala
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (5)..(5)
<223> OTHER INFORMATION: Pro, Ala, Gln, Asp, Glu, Lys, Arg or Val
<220> FEATURE:
<221> NAME/KEY: MOD RES
<222> LOCATION: (7) .. (7)
<223 > OTHER INFORMATION: Ser, Asn or Thr
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (8) .. (8)
<223> OTHER INFORMATION: Val, Ala, Arg, Lys, Gly, Ser, Thr or Glu
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (9)..(9)
<223> OTHER INFORMATION: Thr, Ser, Arg or Asn
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (11) ..(11)
<223> OTHER INFORMATION: Gly, Ser or Asn
<220> FEATURE:
<221> NAME/KEY: MOD RES
<222> LOCATION: (12)..(12)
<223> OTHER INFORMATION: Gly, Lys, Arg, Met, Thr, Leu, Asp, Ser or Pro
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (14)..(14)
<223> OTHER INFORMATION: Val, Ile, Met, Thr, His, Ala, Glu, Phe, Lys,
     Arg, Ser, Gln, Trp or Tyr
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (15)..(15)
<223> OTHER INFORMATION: Gln, Ser, Arg, Lys, Tyr or Ala
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (16)..(16)
<223> OTHER INFORMATION: Thr, Phe, Lys, Gln, Ser, Leu, Glu, Met, Asn or
     Val
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (17)..(17)
<223> OTHER INFORMATION: Arg, Ser or Gln
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (18)..(18)
<223> OTHER INFORMATION: Ser, Thr, Val, Arg, His, Glu, Gln, Ala or Ile
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (19) .. (19)
<223> OTHER INFORMATION: Arg or Val
<400> SEQUENCE: 2420
Xaa Xaa Trp Xaa Xaa Cys Xaa Xaa Cys Xaa Xaa Gly Xaa Xaa Xaa
                                    10
Xaa Xaa Xaa Arg
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<210> SEQ ID NO 2421
<211> LENGTH: 11
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
    peptide
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (2)..(3)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (5)..(5)
<223> OTHER INFORMATION: Thr, Ser or Asn
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (6)..(7)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (9) .. (10)
<223> OTHER INFORMATION: Any amino acid
<400> SEQUENCE: 2421
Trp Xaa Xaa Cys Xaa Xaa Xaa Cys Xaa Xaa Gly
               5
<210> SEQ ID NO 2422
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2422
Ser Ala Pro Phe Ile Glu Cys His Gly Arg Gly Thr Cys Asn Tyr Tyr
                                 10
1 5
Ala Asn Ala
<210> SEQ ID NO 2423
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2423
Ser Ala Pro Phe Ile Glu Cys His Gly Arg Gly Thr Cys Asn Tyr Tyr
Ala Asn Ser
<210> SEQ ID NO 2424
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2424
Ala Thr Pro Phe Ile Glu Cys Asn Gly Gly Arg Gly Thr Cys His Tyr
1
                                   10
Tyr Ala Asn
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<210> SEQ ID NO 2425
<211> LENGTH: 19
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2425
Ala Thr Pro Phe Ile Glu Cys Ser Gly Ala Arg Gly Thr Cys His Tyr
Phe Ala Asn
<210> SEQ ID NO 2426
<211> LENGTH: 19
<212> TYPE: PRT
<213 > ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
     peptide
<400> SEQUENCE: 2426
Ala Ala Pro Phe Leu Glu Cys Gln Gly Arg Gln Gly Thr Cys His Phe
                                    10
Phe Ala Asn
<210> SEQ ID NO 2427
<211> LENGTH: 20
<212> TYPE: PRT
<213> ORGANISM: Artificial Sequence
<220> FEATURE:
<223> OTHER INFORMATION: Description of Artificial Sequence: Synthetic
peptide <220> FEATURE:
<221> NAME/KEY: MOD_RES
<222> LOCATION: (1)..(2)
<223> OTHER INFORMATION: Any amino acid
<220> FEATURE:
<221> NAME/KEY: MOD_RES
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<223 > OTHER INFORMATION: Any amino acid
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Xaa Xaa Xaa Xaa

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Gln Ala Gln His Tyr Val Cys Met
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Gln Ala Lys Phe Phe Ala Cys Ile
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Val Cys Asn Phe
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Val Cys Asn Phe
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<212> TYPE: PRT
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Ile Ile Glu Lys Met Leu Asn Ser
           20
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What is claimed is:

1. An isolated peptide or analog thereof consisting of a sequence having at least 85% amino acid sequence identity to:

Chorionic somatomammotropin or

(SEQ ID NO: 2294)

LLRLLLIESWLE,

(SEQ ID NO: 2295)

Chorionic somatomammotropin

LLHISLLIESRLE

hormone-like 1

wherein the peptide reduces blood vessel formation in a cell, tissue, or organ, and comprises at least one modification

2. The isolated peptide of claim 1, wherein the modification is a sequence alteration or post-translational modification that increases protease resistance, biodistribution, or therapeutic efficacy.

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3. A pharmaceutical composition comprising an effective amount of the isolated peptide of claim 1 in a pharmacologically acceptable excipient.

**4**. A kit comprising an effective amount of the peptide of claim **1**, and directions for using the peptide to treat a disease characterized by undesirable or excess angiogenesis.

**5**. A method of reducing blood vessel formation in a tissue or organ, the method comprising contacting an endothelial cell, or a tissue or organ comprising an endothelial cell, with an effective amount of the peptide of claim **1**, thereby reducing blood vessel formation in the tissue or organ.

**6**. A method of reducing blood vessel formation in a tissue or organ the method comprising:

contacting the tissue, or organ with a vector encoding the peptide of claim 1; and

expressing the peptide in a cell of the tissue or organ, thereby reducing blood vessel formation in the tissue or organ.

7. A method for treating a lung carcinoma in a subject in need thereof, the method comprising administering an effective amount of the peptide of claim 1.

\* \* \* \* \*